Ophthalmology in Military and Civilian Casualty Care

Christopher J. Calvano
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Anthony J. Johnson
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Preface

Ophthalmology in Military and Civilian Casualty Care presents the most current thoughts from experts across multiple fields including government, military, medical, and first responder communities. It is intended to serve as a functional guide for management of vision-threatening injuries and conditions commonly encountered in austere, combat, humanitarian, and mass casualty scenarios. We are deeply grateful to our contributors who have provided their opinion and experience for this unifying manuscript, often by writing in between overseas deployments and ongoing crisis response duties.

The excellent paper by COL Mark Reynolds et al. [1] presents a concise summary of clinical practice guidelines for ocular injuries and vision-threatening conditions requiring prolonged field care, and we refer reader to this manuscript for a concise and useable format. This is especially timely given the recent acknowledgment of the end of the golden hour as we move toward more remote areas of engagement where evacuation may be greater than 48–72 hours delayed, if available at all [2]. Pushing information and skills to the tip of the spear is an obvious mitigation strategy, and our desire is to empower all providers to save vision wherever possible.

We have not sought to be encyclopedic but rather to craft a usable handbook with expanded detail where indicated. Not intended to be another ophthalmology trauma textbook, any deficiencies in content perceived by the reader are solely the responsibility of the managing editor (CJC).

The views expressed in this publication are those of the author(s) and do not necessarily reflect the views, policy, or position of the United States Government, Department of Defense, The Department of the Army, The Department of the Navy, the Department of the Air Force, or subordinate commands.

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References

1. Reynolds M, Hoover C, Riesberg J, Mazzoli R, Colyer M, Barnes S, Calvano C, Karesh J, Nurray C, Butler FK, Keenan S, Shackelford S. Evaluation and treatment of ocular injuries and vision-threatening conditions in prolonged field care. J Spec Oper Med. 2017;17(4):115–26.
2. Farr WD. The death of the golden hour and the return of the Future Guerilla Hospital. JSOU Report 17–10, The JSOU Press 2017.

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Part I

Ophthalmic Considerations

History of Military Ophthalmology

1

Andrew S. Pan and Brett W. Davies

Introduction

Before the Revolutionary War, military physicians without specialized ophthalmic training provided care for injuries involving the eyes and orbit. With advances in technology and medicine, specialized training programs for ophthalmology were established. In 1896, The American Academy of Ophthalmology and Otolaryngology (AAOO) was founded as a distinct medical association for ophthalmologists and otolaryngologists. In 1979, The American Academy of Ophthalmology (AAO) split from the AAOO, establishing ophthalmology as a separate specialty.

With advances in warfare, especially after World War II, the US military recognized the value of physicians trained in the field of ophthalmology. Military residency programs were established to train ophthalmologists with an emphasis on ocular and orbital trauma. These ophthalmologists were trained specifically to provide primary ophthalmic care in theater and definitive care in established evacuation hospitals. Advances in medical equipment and technique improved prognosis after traumatic injury. Those advances include microscopes with sharper optics, antibiotics with better ocular penetration, and advanced suturing techniques. Further advances in technology and logistics in medical evacuation further improved traumatic ophthalmic care.

Although great strides have been made in military ophthalmology, warfare continues to evolve and change. Military ophthalmologists will have to continue to adapt and innovate. Continued training for military ophthalmologists in both clinical patient care and research will have to continue in both war and peacetime to meet the ever-changing demands.

Revolutionary War

The US Army Medical Corp was established during the American Revolution in 1775. A group of physicians with various training backgrounds were embedded in regiments. The wounded were treated in their respective regiments and companies regardless of the nature of their injuries. Common injuries during the time include musket gunpowder burns to the periocular area. Upon taking command of the Army, General George Washington recommended the Colonial Congress to establish a hospital service for the Army.

Subsequently, on July 27, 1775, the Continental Congress created a medical service for a 20,000-man army and named Dr. Benjamin

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Church of Boston as director general and chief physician [1]. He later became an early advocate for smallpox vaccination [1]. In 1777, General Washington ordered the vaccination of all Continental Army recruits to prevent smallpox. It was the first successful Army-wide vaccination program.

Dr. John Jones, born in Long Island, New York in 1729, established the medical school that later became Columbia University's College of Physicians and Surgeons where he was the first Professor of Surgery [2]. He performed ocular surgery and wrote the first American medical book, *Plain, Concise, Practical Remarks on the Treatment of Wounds and Fractures*, in 1775 [2]. The book became the first surgical field manual to be adopted by the US military [2].

War of 1812

During the War of 1812, the Army created a separate medical department under Dr. James Tilton [3]. In 1818, Congress established a permanent Army medical service, with Dr. Joseph Lovell becoming the Army's first true surgeon general [4]. Lovell ordered Army surgeons to keep weather records and investigate the relation of disease to climate [4]. On February 11, 1847, Congress passed an act that gave military rank to medical officers for the first time [4].

During this time, ocular wounds were treated similarly to wounds on the torso and extremities. With the exception of enucleation, surgery was not typically performed for ocular injuries [5]. In addition, patients with ophthalmia were treated with silver nitrate in a solution described as "lunar caustic in the distilled waters" [6].

American Civil War

Surgeon General Joseph K. Barnes directed the complication of case studies of battlefield injuries sustained during the American Civil War, titled *The Medical and Surgical History of the War of the Rebellion* [7]. Ocular injuries in the Civil War constituted about 0.9% (1190 eyes) of the injuries due to bullet and close hand-to-hand combat [7]. Ophthalmic examination consisted of gross cursory exam of the eye and adnexa without uniform exam of vision [7]. Anesthesia in terms of chloroform and ether was routinely performed during this time; however, topical anesthesia for ocular injuries was yet to be adopted. Between 1861 and 1866 in the Union Army, 84,986 cases of purulent ophthalmia and inflammation of the conjunctiva were reported [8].

The period between 1850 and 1870 is often considered the "golden age of ophthalmology." The development of the ophthalmoscope led to more thorough exams and understanding of ocular physiology. In 1817, the first American eye hospital was established in New London, Connecticut. By the end of the Civil War, eye hospitals were present in New York (1820), Boston (1824), Philadelphia (1821, 1822, 1834), and Cincinnati (1827) [7]. These established hospitals led to advances in cataract and glaucoma surgery.

Ophthalmology was established as a full-time specialty in the United States during the American Civil War. In 1863, the American Medical Association recognized it as a distinct specialty. The American Ophthalmology Society was established in 1864 and the American Academy of Ophthalmology and Otolaryngology in 1896. In 1893, Army Surgeon General George M. Sternberg established the Army Medical School and offered specialized training in ophthalmology. After the American Civil War, military physicians became better trained in the medical and surgical treatments of ophthalmology.

World War I

Ophthalmic injuries were present in 2% of all combat casualties with an increasing trend during the static phase of trench warfare [9]. Gravel from sandbags caused by motor and grenade explosions often caused intraocular injuries. X-ray localization and Haab magnets were used

to locate and remove intraocular foreign bodies through scleral incisions [10].

The advent of chemical warfare provided challenges for military ophthalmologists. Mustard gas used by the Germans created chemical injuries to the eye and adnexa. Acute cases were treated with solutions of boric acid or sodium bicarbonate to balance the pH [11]. Before the advent of penicillin, syphilis was rampant and a frequent cause of uveitis.

World War II

After World War I, the importance of specialized medical training was realized. Army programs continued to provide specialized training in ophthalmology and continued to ensure proper training and readiness of military ophthalmology. *The Manual of Therapy: European Theatre of Operations* outlined the fundamental policies for ophthalmic injuries were in the hands of every medical officer before the D-Day invasion [12]. From time to time, letters addressing special conditions, such as keratoconjunctivitis, employment of conjunctival flaps, and intraocular foreign body removal, were sent out from the Chief Surgeon's office [12]. Military physicians would perform eyelid laceration repairs, conjunctival flaps for missing eyelids, and open globe repair to include excision of uveal contents [12]. Since sympathetic ophthalmia does not typically presents until 2 weeks after injury, enucleations were performed generally after evacuation from forward operating areas.

In 1944, Captain Stanley F. Erpf, Dental Corps, while working at the Army Dental Section with the 30th General Hospital in England, pioneered a prosthetic eye from acrylic plastic [10]. The prosthetic eye improved cosmetic outcomes after enucleation and prevented further contracture of the orbit. The US Army produced approximately 10,000 prosthetic eyes during World War II [10]. After the functional loss of an eye, the military would send visually impaired soldiers to specialized rehabilitation centers.

Korean War

The military's need for specialty-trained physicians continued after World War II. To meet the shortage, the US military established residency programs in 1946. Army training hospitals provided the necessary training for specialized board-certified physicians with an emphasis on trauma. Ophthalmology residency programs were started at Brooke Army Medical Center (Texas), Walter Reed Army Medical Center (Washington DC), Fitzsimons Army Hospital (Colorado), and Letterman Army Hospital (California). Seventeen board-certified ophthalmologists were in active duty by the start of the Korean War [13].

Ophthalmologists were assigned to both established hospitals in Korea and Japan and also to mobile army surgical hospitals (MASH) in Korea. Improvement in transportation in helicopters allowed for more timely evacuation of casualties to MASH or established hospitals for definitive care by board-certified ophthalmologists.

Vietnam War

Besides improved suturing technique, improved antibiotics, and emphasis on sterile technique, there has been little change in ophthalmic care since World War II [14]. However, by the 1960s and during the time of the Vietnam War, there had been substantial application of modern technology in ophthalmology. Phacoemulsification for cataracts and vitrectomy systems were developed. Lasers of differing wavelengths were used to treat various conditions. Corneal grafts and glaucoma surgery were performed.

During the Vietnam War, projectiles from landmines, booby traps, grenades, and shells produced the most non-lethal injuries. An ocular injury rate of 9% (in contrast to 0.5% during the American Civil War) was observed [14]. The injuries led to a 50% chance that the injury would lead to the loss of an eye [15].

After first aid in the field, helicopter transportation to field and evacuation hospitals

significantly shortened transport time. However, the high volume (estimated 37,700 total injuries to the eye and adnexa) and high incidence of ocular injury created equipment and staff shortages [15]. One ophthalmologist was assigned to each evacuation or field hospital [15]. Many of the nurses and technicians working with the ophthalmologists were not familiar with ophthalmic surgical techniques. There were also not enough fully trained ophthalmologists to staff every evacuation hospital. Most ophthalmologists who served in Vietnam had either just completed their residency training and not board certified (71%) or were not fully trained (21%) [15]. Only 7% of ophthalmologists who served in Vietnam were board certified [15]. These shortages necessitated the evacuation of many patients back to the Communication Zone in Japan and the Philippines. Despite the shortcomings in personnel and equipment, no documented cases of sympathetic ophthalmia were reported [15].

During this period, ultrasound techniques were developed to localize intraocular foreign bodies though opaque media and field tests of polycarbonate eye protection were performed. The soldiers typically did not wear eye protection, as eye protection was not mandated. It was later concluded that 39% of the ophthalmic injuries in Vietnam could have been avoided with eye protection [16].

After the Vietnam War, the Army decided to mandate polycarbonate, the material of choice for eye protection, and to incorporate laser protection into protective eyewear [16]. Further cooperation with civilian eyewear manufactures led to continued improvements in protective eyewear.

Afghanistan and Iraq (Operation Enduring Freedom and Operation Iraqi Freedom)

Since the Vietnam War, the US Army has been involved with several conflicts around the world: in Panama, Grenada, Haiti, Somalia, the Persian Gulf, and the Balkans. Military ophthalmic care was especially important during the Persian Gulf War, as Army and Navy ophthalmologists provided care for 221 casualties [17].

Operations in Afghanistan and Iraq presented unique challenges for military ophthalmologists as the militants utilized unconventional warfare against the US military. The militants incorporated improvised explosive devices (IEDs) into their unconventional warfare. Advancements in modern armor better protected the soldiers' chest and abdominal area from blast injuries, but left the face and eyes relatively unprotected. Twenty-two percent of the casualties from these conflicts who passed through the military's Landstuhl Regional Medical Center in Germany had injuries to the head, face, or neck [18]. From 2002 through 2007, the number of US military OIF/OEF soldiers with significant ocular injuries requiring evacuation was 13% [19].

From 2003 to 2006, of the casualties evacuated to Walter Reed Army Medical Center sustaining globe or adnexal injuries, 26% had penetrating (open) globe injuries, 22% had non-penetrating (closed) globe injuries, 36% oculoplastic injuries, and 15% had injuries to the brain/optic nerve [19]. The vast majority of injuries were from blast injuries (79%), followed by gunshot wounds (9%), and motor vehicle accidents (8%) [19].

The emerging Afghanistan battlefield had numerous forward surgical teams, combat support hospitals, and medevac assets from all three services. The secretary of defense, Robert Gates, maintained that serious casualties should be evacuated in 60 minutes or less [19]. Secretary Gates believed that the Golden Hour doctrine is not only vital in providing excellent medical care but was a key component of troop morale and expectations [19]. He directed additional resources (25 medevac helicopters and 3 forward surgical hospitals) to Afghanistan [20]. After Secretary Gates' mandate, aerial evacuation times fell from 90 minutes to 43 minutes in Afghanistan (with mortality decreasing from 16% to 9.9%) [20].

Improvements in aerial evacuations allowed level-three hospitals to provide intermediate care and speedily send casualties to more definitive

care in Germany and the United States. For example, a solider may suffer a penetrating globe injury in theater. The globe would be anatomically closed at a local combat support hospital and sent back to a military hospital stateside for further surgical care (such as facial reconstruction, retinal detachment repair, and anterior segment reconstruction) within 36 hours. The speedy surgical care provided excellent visual outcomes. The visual acuity at 6-month follow-up improved in 28% of patients with globe injuries, and 42% of eyes achieved a best-corrected visual acuity of 20/40 or better [20].

Implications for Military Ophthalmologists

The US Army Medical Department currently has training centers for ophthalmologists at three Army Medical Centers: San Antonio Military Medical Center (San Antonio, TX), Walter Reed National Military Medical Center (Bethesda, MD), and Madigan Army Medical Center (Tacoma, WA). In addition, further subspecialty-training fellowships and postgraduate courses with focus on ocular trauma are offered to military ophthalmologists every year.

Presently, Army ophthalmologists must be prepared for both combat and non-combat missions. In addition to supporting combat operatives of US servicemen and allies around the world, Army ophthalmologists must be ready to provide humanitarian and civic assistance. Resources are often scarce in deployed situations; therefore, the Army ophthalmologist must be resourceful and prepared to provide surgical care outside the scope of ophthalmology. With the ever-changing geo-political environment, the Army ophthalmologist must be ready to adapt and innovate in order to provide the best care possible.

References

1. Loughlin KR. Benjamin church: physician, patriot, and spy. J Am Coll Surg. 2001;192(2):215–9.
2. Griesemer AD, Widmann WD, Forde KA, et al. John Jones, M.D.: pioneer, patriot, and founder of American surgery. World J Surg. 2010;34(4):605–9.
3. Tilton FJ. History of the Tilton family of America, Vol. I (1928).
4. Craig SC. Joseph Lovell, MD (1788–1836): first US army surgeon general. J Med Biogr. 2016;24(3):309–19.
5. Ashburn PM. History of the Medical Department of the US Army. Houghton Mifflin: Boston; 1929.
6. Melin GR. The Army Medical Department, 1818–1865. Am Med Rec. 1825;8:193.
7. Hertle RW. Ophthalmic injuries and civil war medicine. Doc Ophthalmol. 1997;94(1–2):123–37.
8. Otis GA, Huntington DL. Medical and surgical history of the war of the rebellion. Washington, D.C.: US Government Printing Office; 1883.
9. Vail DT. Military ophthalmology. Trans Am Acad Ophthalmol Otolaryngol. 1951;55:709–15.
10. Thach AB. Ophthalmic care of the combat casualty. Falls Church/Washington, D.C./Fort Sam Houston/Bethesda: Office of the Surgeon General, United States Army Borden Institute, Walter Reed Army Medical Center United States Army Medical Dept. Center and School Uniformed Services University of the Health Sciences For sale by the Supt. of Docs., U.S. G.P.O; 2003. Print.
11. Patterson RU. Address of the surgeon general. Presented at: annual meeting of the American Academy of Ophthalmology and Otolaryngology. September 16, 1933. Boston.
12. Coates JB. Medical Department United States Army in World War II: ophthalmology and otolaryngology. Washington, D.C.: Office of the Surgeon General Department of the Army; 1986.
13. King JH. Army ophthalmology, past and present. Mil Surg. 1953;112:88–96.
14. Biehl J. Penetrating eye injury in war. Mil Med. 1999;164(11):780.
15. La Piana FG, Hornblass A. Military ophthalmology in the Vietnam War. Doc Ophthalmol. 1997;93:29–48.
16. Ari AB. Eye injuries on the battlefields of Iraq and Afghanistan: public health implications. Optom J Am Optom Assoc. 2006;77(7):329–39.
17. Mader TH, Aragones JV, Chandler AC, et al. Ocular and ocular adnexal injuries treated by United States military ophthalmologists during Operations Desert Shield and Desert Storm. Ophthalmology. 1993;100:1462–7.
18. Okie S. Traumatic brain injury in the war zone. N Engl J Med. 2005;352:2043–7.
19. Weichel ED, Colyer MH, Ludlow SE. Combat ocular trauma visual outcomes during operations Iraqi and enduring freedom. Ophthalmology. 2008;115(12):2235–45.
20. Farr WD. The death of the golden hour and the return of the future guerrilla hospital. MacDill Air Force Base: The JSOU Press; 2017.

Damage Control Ophthalmology

2

Anthony J. Johnson and J. Richard Townley III

Introduction

The concept of damage control surgery was developed by trauma surgeons as a methodology to focus the initial trauma surgery effort, after significant injury, toward dealing with the trauma triad of death: coagulopathy, acidosis, and hypothermia. Thus, their initial surgical effort emphasizes physiologic recovery over anatomic reconstruction [1]. As ophthalmologists performing surgery in times of conflict on one of the most important sense organs, that of sight, with triage imperatives of protecting life, limb, and eyesight, we may be required to operate alongside and sometimes simultaneously with our trauma surgeons during this damage control period. Given that >70% of ruptured globes during our most recent military conflicts were associated with significant systemic injuries, we have by necessity and doctrine been performing our own version of damage control surgery for decades [2].

Due to the unique physiology and anatomy of eye structures, our approach to damage control ophthalmic surgery is by necessity very different from our trauma colleagues who delay anatomic reconstruction in an effort to restore physiologic function [1]. Like our trauma colleagues, our approach involves careful surgical planning with repair of critical anatomic structures in a staged approach, which if adhered to results in optimum outcomes for the patients.

Critical to this process is an understanding of the anatomic and physiologic processes of the eye. Repair of a seriously injured eye should not be undertaken unless the physician understands the anatomy, physiologic, and pathophysiology of trauma; can properly classify the injury; and knows the unique features of each injury type [3]. Additionally, to optimize outcomes, the damage control ophthalmology principles should be implemented within the framework of a larger trauma designed to protect the eye from point of injury, through the chain of evacuation, to the ophthalmic surgeon. In this chapter, we cover the basic framework of damage control surgery. The following chapters discuss aspects of the surgery in greater detail.

Terminology

To understand the principles of damage control ophthalmology and to define the prioritization of effort, we need a common nomenclature to describe injuries to the eye. The University of

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Alabama Birmingham developed a nomenclature that has been accepted by the American Academy of Ophthalmology and the International Ocular Trauma Society [4]. Central to the nomenclature is the concept that all definitions refer to the entire globe and not individual tissues. If a tissue is mentioned, it is done so to qualify the location of the injury. Table 2.1 outlines the agreed-upon terminology for ocular injuries. Figure 2.1, from the Birmingham original publication, shows graphically how this terminology is employed. Figure 2.2 utilizes a decision tree to assist the provider in determining the correct terminology.

Table 2.1 Table of terminology [3]

Term	Definition	Comment
Eye wall	Sclera and cornea	Though the eye wall has three layers posterior to the limbus, clinical and practical purposes dictate that violation of only the most external tissue (sclera) is to be considered
Closed globe injury	No full-thickness wound of eye wall	The cornea and the sclera are not breached through and through
Open globe injury	Full-thickness wound of the eye wall	The cornea and/or sclera is breached through and through
Contusion	No wound of the eye wall	The damage may be due to the direct energy delivery/shock wave by the object (i.e., choroidal rupture), or to changes in the shape of the globe (i.e., angle recession)
Lamellar laceration	Partial-thickness wound of the eye wall	The wound in the eye wall is not "through" but "into"
Rupture	Full-thickness wound of the eye wall, caused by a large blunt object	Since the eye is filled with incompressible liquid, the impact results in instant *intraocular pressure* (IOP) elevation. The eye wall yields at its weakest point (rarely at the impact site, rather, for instance, along an old cataract wound); the actual wound is produced by an inside-out mechanism, and tissue prolapse is almost unavoidable
Laceration	Full-thickness wound of the eye wall, caused by a sharp object	The wound is at the impact site and is created by an outside-in mechanism; since *intraocular pressure* (IOP) elevation is unavoidable, tissue prolapse is common
Penetrating injury	An entrance wound is present	If more than one wound is present, each must have been caused by a different object
Intraocular foreign body (IOFB)	One or more foreign objects are present	Technically a penetrating injury, but grouped separately because of different clinical implications (management, prognosis)
Perforating injury	Both an entrance and an exit wound are present	The two wounds caused by the same agent

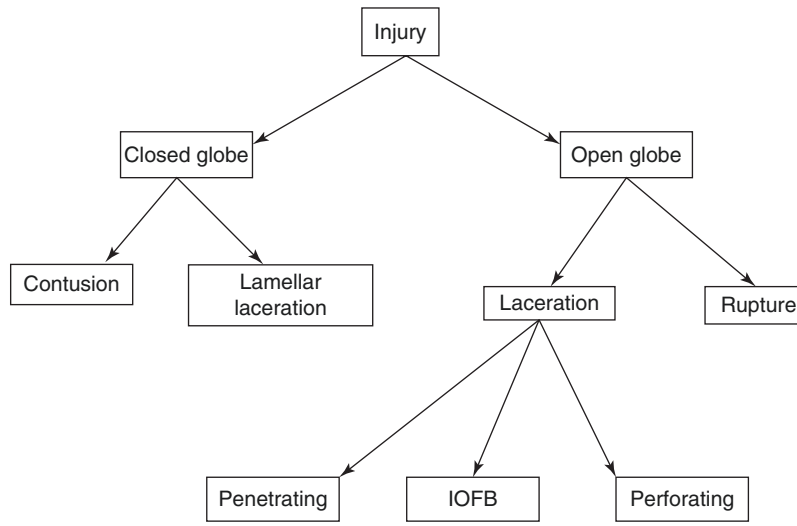

Fig. 2.1 Graphic representation of employment of the terminology of eye injuries [4]

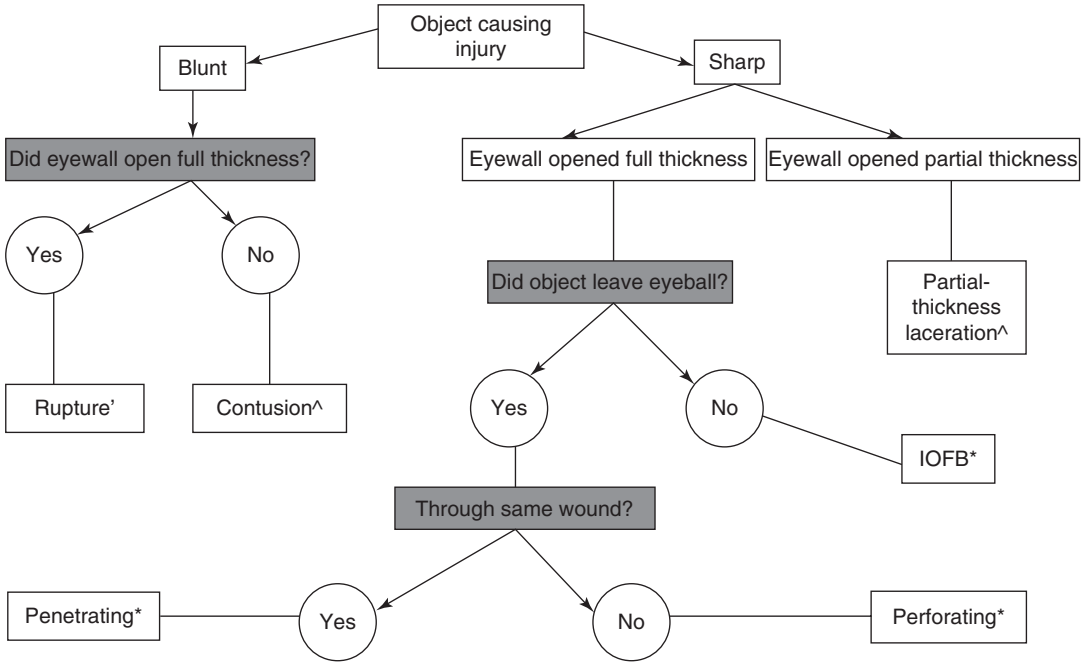

Fig. 2.2 Decision tree to assist the provider in determining the correct terminology. Injuries marked with asterisk (*) are open globe injuries. Injuries marked with caret (^) are closed globe injuries [4]

Ocular Trauma Score (OTS) has been devised to assist in communicating with patients regarding the likelihood of visual recovery after surgery.

Ocular Trauma Score [5]

Through the evaluation of 2500 trauma patients, the Ocular Trauma Score was developed to reliably predict the functional outcome of serious eye injuries. The Ocular Trauma Score is easy to calculate: The patient is given initial points for their initial vision. Then points are deducted for the presence of a globe rupture, endophthalmitis, perforating globe injury, retinal detachment, or afferent pupillary defect, according to a standardized value for each (Table 2.2).

The final raw points are then converted into an OTS, and Table 2.3 gives the likelihood of the final visual acuity separated into five categories.

By employing the information given by the OTS, we can now counsel our patients and more predictably engage them in the triage, management, and rehabilitative process they can expect as a result of their injury.

Table 2.2 Calculating the Ocular Trauma Score (OTS): variables and raw points [5]

Variable	Raw points
Initial vision	
NLP	60
LP/HM	70
1/200–19/200	80
20/200–20/50	90
≥20/40	100
Rupture	−23
Endophthalmitis	−17
Perforating injury	−14
Retinal detachment	−11
Afferent papillary defect	−10

Triaging Eye Injuries

Critical to the proper employment of damage control ophthalmic principles, the process of triaging injuries is paramount. There is no delayed primary closure of open globes. Open globes

Table 2.3 Calculating the OTS: conversion of raw points into an OTS category, and calculating the likelihood of the final visual acuity in five categories [5]

Sum of raw points	OTS	No light perception (%)	Light perception/hand motion (%)	1/200–19/200 (%)	20/200–20/50 (%)	≥20/40 (%)
0–44	1	74	15	7	3	1
45–65	2	27	26	18	15	15
66–80	3	2	11	16	31	41
81–91	4	1	2	3	22	73
92–100	5	0	1	1	5	94

should be closed as quickly as possible in an effort to prevent choroidal bleeding, infection, and epithelial downgrowth.

The open globe is always at risk for expulsive hemorrhage, in which bleeding from underneath the retina forcefully pushes all the intraocular structures out of the eye, causing total blindness. The normal intraocular environment is an intraocular pressure (IOP) of 15–20 mm Hg above atmospheric pressure. When the eye is open, the pressure drops to 0 mm Hg. The retinal and choroidal vasculature are anteriorly displaced placing traction of the vascular plexus where they are tethered at the intrascleral canals [6]. Additionally, the low intraocular pressure exacerbates the transmural forces across the vascular structures, shifting the Starling forces toward accumulation of fluid in the extravascular space (choroidal effusion) [6]. These forces are exacerbated if the patient suffered from pre-injury elevated intraocular pressure or elevated episcleral venous pressure such as would occur with glaucoma. If the injured patient was older, suffered from atherosclerotic vascular disease, heart disease, hypertension, obesity, or diabetes (all of which can increase the fragility of their vascular system), their risk of intraocular bleeding is elevated. This risk slowly increases the longer the eye is opened.

In the absence of a direct wound or intraocular foreign body, the risk of infection does not measurably rise if the globe is closed within 24 hours [7]. The only exception is when the globe cannot be successfully closed or has endured so much damage that sight is not possible. In those cases, enucleation or evisceration should be entertained. If enucleation is to be performed, every effort should be made to include the patient in the conversation.

As is evident by the ocular trauma score, visual acuity is very important in triaging eye injuries. In general, the worse the presenting visual acuity, the worse the final visual outcome.

General Principles of Damage Control Ophthalmic Surgery

Open globes should be closed as quickly as possible in a water-tight fashion (to prevent expulsive hemorrhage, infection, or epithelial downgrowth). There is no delayed primary closure. After closure is performed, verification of water-tight status is performed via the Siedel test.

- When confronted with a complicated multiple-wound globe trauma, it can be challenging to know where to begin. All patients undergoing repair therefore should be under general anesthesia to allow for full and careful exploration of the extent of their injuries without causing any additional trauma to the intra and extraocular structures.
- Any foreign bodies protruding through the lids, cornea, or conjunctiva should be stabilized and not removed until it can be fully investigated.
- Always work from the known to the unknown. To do this, you need to ensure you have adequate exposure by carefully retracting the eyelids using specialized lid retractors that are developed to take pressure off the globe such as the Jaffe or Schott lid speculums. If those are unavailable or the injury to the lids is not amenable to accommodating the proper speculums, you may consider using a temporary

stay suture in the upper and lower tarsus to retract the lids up and away from the globe.

- Once the eye is adequately exposed, carefully begin identifying structures that are easily recognizable such as areas of the limbus. Generally, it is best to work anterior to posterior so that intraocular contents will not be inadvertently expressed while trying to reach more posterior wounds.
- If possible, administer broad-spectrum intravenous (IV) antibiotics as close to time of injury as possible.
- The globe should be closed before ocular manipulations are performed (such as forced ductions).
- If there is a full-thickness lid laceration present, one should rule out the presence of an open globe beneath.
- Use nylon sutures, especially if future reconstructive surgery is anticipated in the immediate future.
- A 360-degree peritomy should be performed starting from the unaffected side slowly working your way to the involved area of the globe, being careful not to exert any undue pressure on the globe.
- Close limbus first with 9-0 nylon surgery, close cornea next with 10-0 nylon sutures, then close the sclera with 8-0 nylon suture. Close as far back as can be performed without causing undue pressure on the globe.
- In general, do not excise cornea tissue, ciliary body, choroid, or retina (unless grossly contaminated).
- After all visible and easily accessible wounds have been closed, all four quadrants should be explored, taking care to isolate all four rectus muscles and search underneath and posterior to their insertions to ensure there are no other hidden injuries.
- One to two rectus muscles can be disinserted from the globe using standard strabismus surgery techniques to ensure the muscle will not be lost and can be easily reinserted. Disinsertion may allow for better visibility and exposure of more posterior wounds.
- Very posterior wounds that cannot be safely reached should be left to close on their own (they typically fibrose and close within 1 week).
- Simple interrupted sutures are preferred. Complicated lacerations may require mattress or running sutures.
- Iris and retinal tissue should be reposited unless it is contaminated, necrotic, or has been out of the eye for >24 hours.
- Vitreous outside the wound should be excised via Wexcell or vitrector (vitrector is preferred); care should be taken to not pull on the vitreous as it can exacerbate intraocular injuries.
- Hyphema should be evacuated in the acute setting. It should be performed only if the IOP cannot be controlled or there is evidence of corneal blood staining.
- Lens injuries do not necessarily have to be addressed immediately (early removal can facilitate visualization of the posterior segment).
- Intraocular lens implantation should be delayed until there are no signs of infection or posterior segment pathology.
- Due to the risk of sympathetic ophthalmia, all open globes should be closed. If it is not possible, then it should be excised within 2 weeks with complete removal of all uveal tissue.
- If there are adnexal injuries around the eye, always check for canalicular laceration; these should be stented with a suitable stent as soon as possible.
- Confirmed intraocular foreign bodies should be removed immediately (possible exceptions—non-vegetative, non-toxic intraocular foreign bodies (IOFBs) from blast injuries can undergo delayed removal).
- Non-sutured posterior perforating injuries take 1 week to scar and stabilize and will not receive immediate surgery. (This should be taken into consideration when arranging aero-evacuation in potentially high-risk environments.)
- Many small intraorbital foreign bodies can remain in the orbit without much consequence. Therefore, if a foreign body is not easily accessible or amenable to safe removal at the time of the initial closure, it may remain in the orbit to be dealt with at a later time if necessary.

References

1. Schwab CW. Introduction: damage control at the start of 21st century. Injury. 2004;35(7):639–41.
2. Thatch AB, Johnson AJ, Carroll RB, et al. Severe eye injuries in the war in Iraq, 2003–2005. Ophthalmology. 2008;115(2):377–82.
3. Kuhn F, Morris R, Mester V, Witherspoon CD. Ocular traumatology. In: Kuhn F, editor. Terminology of mechanical injuries: the Birmingham Eye Trauma Terminology (BETT). Cham: Springer; 2008. p. 3–11.
4. Kuhn F, Morris R, Witherspoon CD. Birmingham Eye Trauma Terminology (BETT): terminology and classification of mechanical eye injuries. Ophthalmol Clin North Am. 2002;15(2):139–43.
5. Kuhn F, Maisiak R, Mann L, Mester V, Morris RS, Witherspoon CD. The ocular trauma score (OTS). Ophthalmol Clin North Am. 2002;15(2):163–5.
6. Kuhn F, Slezakb Z. Damage control surgery in ocular traumatology. Injury. 2004;35(7):690–6.
7. Eiseman A, Birdsong R, editors. Ocular trauma course syllabus. Bethesda: Uniformed Services University of the Health Sciences; 2009.

3 Damage Control Ophthalmology: Emergency Department Considerations

Ronnie K. Ren and Daniel J. Dire

Introduction

Two percent of emergency department (ED) patients have eye complaints including trauma and infections, and eye injuries account for 3.5% of all occupational injuries in the United States [1]. Most of these patients are treated by the emergency physicians with or without consultation with an ophthalmologist. The eyes represent 0.1% of the total body surface area; yet, they account for 8–13% of battle injuries [2]. On the battlefield, all eye injuries should be considered significant because even a small corneal abrasion can incapacitate a soldier.

The incidence of eye injuries ranged from 0.57% in the American Civil War, 1.5–3.8% in World War I and II, 5–9% in the Vietnam War, to 13% in the first Persian Gulf War in 1991. During OIF and OEF from March 2003 to December 2004, 17.8% of all medical evacuations were for battle or non-battle eye injuries. The major causes (70%) of these eye injuries were IEDs, RPGs, and shrapnel, many occurring during convoy operations. The increase in combat-related eye injuries is due in part to the increased survivability of the wounded, the lack of use of eye protection, and the increase in blasts containing small fragments [3].

The majority of modern combat ophthalmic injuries are associated with concomitant non-ocular life-threatening injuries [4]. Often the initial eye examination is performed in the operating room after surgical stabilization [5].

Emergency Approach to the Field Ophthalmologic Conditions

The goal for emergency ophthalmologic care is twofold. First, one must provide care that preserves a patient's visual function and cosmetic appearance. This involves adequate initial care and appropriate referral to an ophthalmologist. Second, one must identify ocular manifestations of systemic disease and address it accordingly. In general, an ophthalmologic problem can be classified into one of the four cardinal complaints of the eye: (1) change in vision, (2) change in appearance of the eye, (3) discomfort of the eye, and (4) trauma of the eye [6, 7].

In terms of treatment and disposition, the emergency diagnosis of an ocular condition can be further arranged into one of the following three categories [8, 9]:

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1. *Emergent*—requires immediate ophthalmologic consultation and treatment. This includes sudden vision loss, chemical burns, globe penetration or rupture, and orbital compartment syndrome.
2. *Urgent*—requires ophthalmologic follow-up within 1 day or less, and much of emergent eye care can be performed by the emergency physician. This includes acute glaucoma, orbital cellulitis, hyphema, macular edema, retinal detachment, and corneal abrasion or ulcer.
3. *Non-urgent*—ophthalmologic follow-up within 2 days. This does not exclude other emergent medical conditions where an ocular symptom was the presenting complaint. This includes conditions such as methanol poisoning, occipital stroke, and cavernous sinus thrombosis.

Initial Approach

In approaching any patient with an ocular complaint, one must remember there are few absolute ophthalmologic emergencies where treatment and subsequent consultation can be (and should be) initiated with only initial history and inspection. Any emergent, non-ocular condition that may compromise safety or patient's airway, breathing, and circulation must always be addressed first prior to any ophthalmologic evaluation. Thus, combat damage control surgery in an FST/FSRT or field hospital may take precedence over the initial eye evaluation.

If a patient has clear history and appearance of globe penetration or rupture, it is an absolute ophthalmologic emergency [9, 10]. Stabilize (if large), but do not remove, any visible foreign body (Fig. 3.1). Place a protective eye shield over the eye without applying any pressure to the globe. Elevate head of the bed to 30° or higher. Experts recommend prophylactic antibiotics as it theoretically helps prevent endophthalmitis. Suggested antibiotics are vancomycin (15 mg/kg, max 1.5 g) plus ceftazidime (50 mg/kg, max 2 g) or alternately, a fluoroquinolone in patients allergic to penicillin. This will cover organisms commonly associated with post-traumatic endophthalmitis such as Bacillus species, coagulase-negative Staphylococcus, Streptococcal species, *S. aureus*, and gram-negative organisms [11–14].

Retrobulbar hemorrhage and orbital compartment syndrome are very rare after ocular trauma or recent surgery, but highly sight threatening. It should be suspected in any patient with pain, decrease in vision, and sign of proptosis. Orbital compartment syndrome is diagnosed by the presence of proptosis, visual acuity deficit, and intraocular pressure (IOP) greater than 40 mmHg, and supported by the presence of an afferent pupillary defect and ophthalmoplegia. The association of blindness with retrobulbar hemorrhage can be as high as 48% but as low as 0.14% if treated promptly and properly [15, 16]. As such, lateral canthotomy with cantholysis should be performed on these patients, ideally within 2 hours of symptom onset [17, 18]. Acetazolamide 500 mg IV may also be given, similar to patients

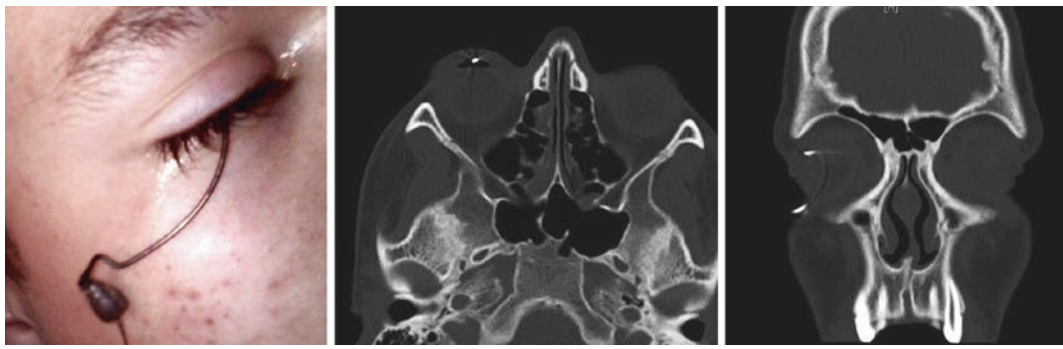

Fig. 3.1 Patient with a fishhook embedded into his right globe with axial and coronal CT images

with acute angle-closure glaucoma. An ophthalmologist should be emergently consulted but treatment should not be delayed for their arrival. For patients with elevated IOP but <40 mmHg, conservative management can be considered, but the provider should maintain a low threshold for canthotomy. Globe rupture is a contraindication to lateral canthotomy.

Chemical exposure to the eye, especially alkalis, acids, or mustard gas, is an absolute ophthalmologic emergency [9, 10, 19–21]. Alkali injuries are more concerning because they cause liquefaction necrosis to the cornea that will rapidly and continuously penetrate the eye until irrigated to a normal pH. Caustic exposures to the eye should be immediately and copiously irrigated with water or a crystalloid solution, if possible, prior to any further evaluation. In the field, potable water from a canteen should be used by the battle buddy until evacuation to the nearest medical treatment facility can occur. In the ED or field medical treatment facility (MTF), first instill a topical anesthetic into both eyes to provide anesthesia to the affected eye and to blunt the blink reflex from the unaffected eye. Next evert the eyelid and sweep out any particulate matter with a moistened cotton-tip applicator. Next retract the eyelids with Desmarres (or paperclip) retractors during irrigation. Alternately, and preferably, a Morgan Lens should be inserted (Fig. 3.2). Irrigate for 30 minutes and measure the pH in the conjunctival cul-de-sac. Repeat irrigation until the eye pH is back to the normal range (7.0–7.4).

Consider complete retinal artery occlusion (CRAO) in a patient with sudden, painless, and complete loss of vision to one eye [8, 9]. In this setting, it is reasonable and not harmful to provide immediate ocular massage, paper bag (CO_2) breathing, and supplemental oxygen prior to proceeding with further evaluation and emergent ophthalmology consult.

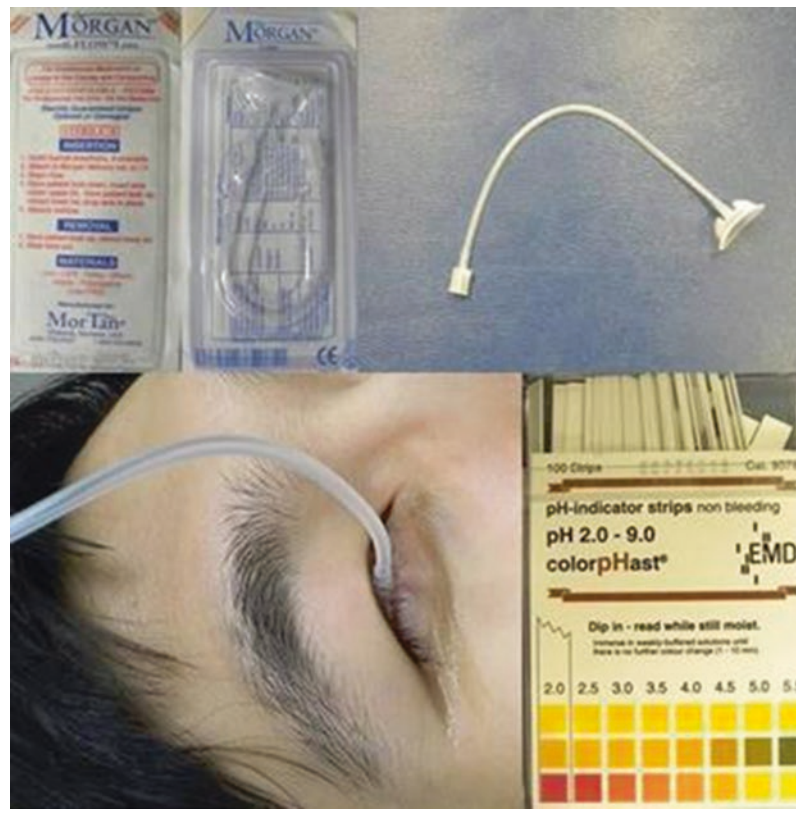

Fig. 3.2 Morgan Lens, MorTan® Inc., Missoula, MT can be connected to IV bag of normal saline or Ringer's lactate for continuous irrigation. pH strips are used to measure the eye pH after each 30 minutes of irrigation

Ophthalmologic History

Classify the chief complaint and get descriptors: (1) onset, (2) provocation (aggravating and relieving factors), (3) quality, (4) radiation and associated symptoms (e.g., headache, nausea, and vomiting in acute glaucoma), (5) severity, (6) time (acuity and progression), (6) visual changes (monocular vs. binocular, painful vs not painful, diplopia, photophobia).

A patient's past medical history may yield useful information. It is important to know if a patient wears corrective lenses at baseline. Contact lens wearers are at increased risk for corneal injury and if so, must be covered for pseudomonal infections. Ocular procedures cannot be performed with contact lenses still in place. Certain ophthalmologic medications and procedures may affect the ocular exam, particularly the pupillary response. The most common cause of irregular or non-reactive pupil is previous ocular surgery [22]. Existing ocular disease such as cataracts or glaucoma significantly affects a patient's baseline and perception of acute injury. For example, a person with significant cataract in one eye might perceive acute deficit in his other, healthy eye as binocular vision loss. Lastly, systemic disease can also put patients at risk for ocular disease. Hypertension and diabetes are leading causes of retinopathy [9]. Patients with HIV develop many ocular pathologies as well which is beyond the scope of this textbook.

Lastly, determine the patient's tetanus vaccination history and provide tetanus vaccinations as indicated [23].

Developing a Differential Diagnosis

In approaching an ocular complaint, the provider must realize that eye symptoms are often the first noticed abnormality in many neurologic and other systemic conditions. As such, differentials for eye complaints are wide (Fig. 3.3). It is crucial to obtain detailed history of a patient's eye complaint and take note of features that point to an extraocular pathology.

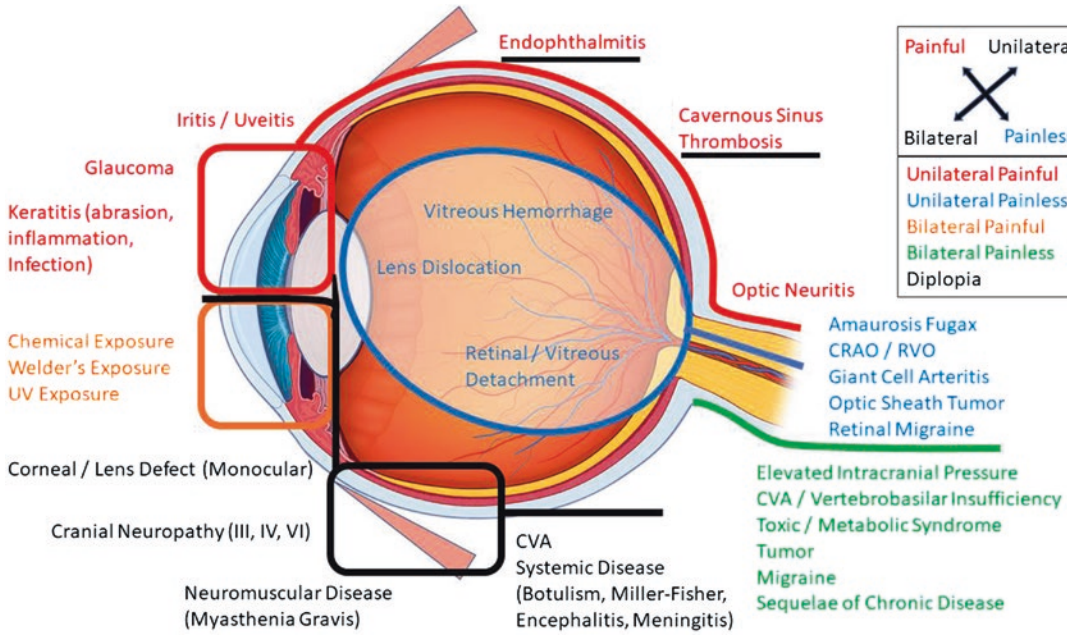

Fig. 3.3 Depiction of the differential diagnosis of ocular disturbances based on painful and painless and unilateral and bilateral visual disturbances

In approaching atraumatic vision disturbance, the provider may take the following steps to develop a differential. First, determine whether the complaint is visual deficit or double vision. Then determine whether it is monocular or binocular. Lastly, determine whether it is associated with eye pain or not.

Monocular painless vision disturbance has a few emergencies. This includes complete vision loss from central retinal artery occlusion and giant cell arteritis. These people tend to be older and have cardiac risk factors. Temporal arteritis is associated with temporal headaches and jaw claudication, but the vision loss component is generally painless. Other less emergent causes include retinal and vitreous detachment as well as hemorrhages. Generally, monocular painless vision loss will have a key finding on fundoscopy, an exam rarely performed by the emergency department or primary care providers. Ocular ultrasound may be helpful (see the following sections).

Binocular painless vision disturbance is almost always neurologic, toxicologic, or metabolic. Acutely, they rarely cause complete vision loss with no light perception. Strokes may cause visual field deficits. Posterior circulation lesions may cause bilateral complete vision loss, but it will be associated with other neurologic findings that take precedence. In patients with low risk for stroke, strongly consider toxicologic causes.

Monocular painful vision disturbance will almost always have a finding on external exam. It is important to note association with trauma, foreign body, chemical exposure, contact use, and other extraocular symptoms. Conditions such as acute angle-closure glaucoma, keratitis, and uveitis will have characteristic ocular exam findings. Other conditions like endophthalmitis, cavernous sinus thrombosis, and optic neuritis will have other extraocular and systemic findings.

Binocular painful vision disturbance can be caused by anything that causes monocular painful vision loss. In addition, the provider must consider chemical exposure, welder's exposure, and ultraviolet light exposure.

Diplopia can also be monocular or binocular. Monocular diplopia does not correct when one eye is covered. This is generally a refractory problem of the cornea or lens. Binocular diplopia corrects when one eye is covered. Its causes are typically structural, neurologic, or metabolic.

In approaching eye discomfort, it is important to note how the symptoms came to be: trauma, foreign body, or irritant exposure. Any cause of painful visual disturbance should be considered. Photophobia could be of ocular etiology such as acute angle-closure glaucoma or uveitis, or neurologic such as meningitis or migraine. Infectious and immunologic causes should be considered.

Ophthalmologic Exam

External Exam

External ocular exam begins the moment the provider sees the patient. This will passively continue for the duration of the evaluation. Few, active steps must then be taken to complete the external exam. The most immediate priority of the external exam is to identify features of significant orbital trauma (Fig. 3.4). This includes globe rupture or penetration, chemical injury, and orbital compartment syndrome. Subsequently, features of urgent ocular conditions will be evaluated, including eyelid trauma, neurologic pathology, infection, and corneal or uveal pathology.

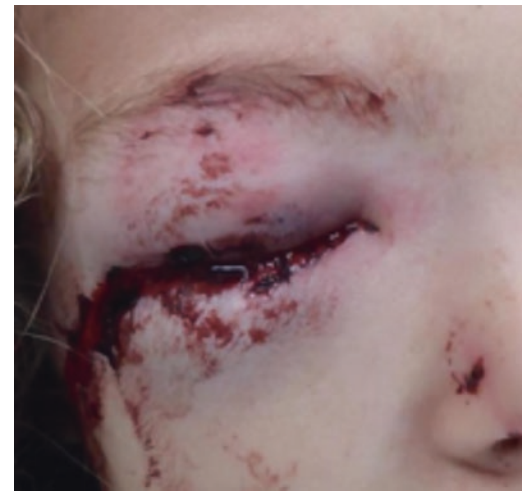

Fig. 3.4 A 3-year-old child with dog bite wound to the eye initially suspicious for penetrating globe injury

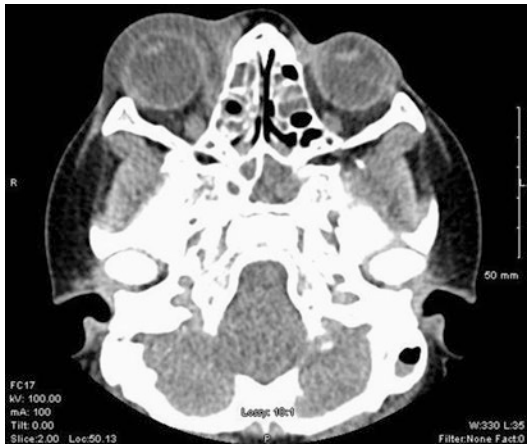

Fig. 3.5 Axial CT image of a 10-year-old child with right orbital cellulitis demonstrating proptosis of the right globe

Fig. 3.6 Hordeolum of the upper lid of the right eye

Start the exam away from the eye and gradually move closer. Observe for proptosis (Fig. 3.5), which may be suggestive of orbital cellulitis or retroorbital hematoma [6, 10], and enophthalmos (sunken eye), which may indicate orbital rupture or a blowout fracture [24]. It is important to note that retroorbital hematoma is by far the most common cause of orbital compartment syndrome. This is an absolute ocular emergency requiring immediate intervention or irreversible vision loss may occur in as little as 2 hours [25]. Subtle findings can be identified by observing the patient from above the head and looking downward toward the eyes [26].

Evaluate the periorbital area including the eyelids. During the exam, it is critical to not palpate any part of the globe if there is suspicion of rupture. Consider orbital wall fracture in setting of decreased infraorbital sensation, palpable step-off around the orbital rim, point tenderness, ecchymosis, or anophthalmos [10, 24, 27]. Evaluate for other signs of trauma such as periorbital swelling or ecchymosis, and lacerations. If a patient has ptosis, one lid will appear droopy and a Horner's syndrome or a third cranial nerve lesion may be present [27].

Localized swelling of the eyelid may be due to a hordeolum or a chalazion. Hordeolum, (Fig. 3.6) a localized infection (most commonly *S. aureus*) of the lash follicle that can cause pain, colloquially known as a "stye" is usually found at the lid's edge [6, 27, 28]. An internal hordeolum involves the meibomian glands, an external hordeolum involves the glands of Zeis or Moll at the base of the eye lashes. Chalazion, a painful swelling of the Meibomian gland of the eyelid, is commonly away from the lid's margin [27, 28]. Trichiasis may cause an eyelash to turn inward, rubbing against the cornea and causing foreign body sensation [6, 20].

To examine the inner lower eyelid, pull down the lower lid from inferior orbital rim and have the patient look up. This will allow examination of the inner lower eyelid to look for foreign body. To see the inner aspect of the upper lid, one must evert the lid (Fig. 3.7). Do not evert the lid if rupture of the globe is suspected [20, 21, 24, 29]. Subconjunctival hemorrhage can be spontaneous or secondary to trauma [24]. It is usually harmless but if it encircles the entire cornea and is associated with trauma, globe rupture should be considered [21, 28].

Conjunctival injection can be classified into conjunctival, limbal, and mixed. Conjunctival injection without limbic involvement is typically caused by superficial pathologies like conjunctivitis. When there is limbic injection or ciliary flush (area immediately surrounding the cornea is inflamed), pathologies of the cornea, uvea, and

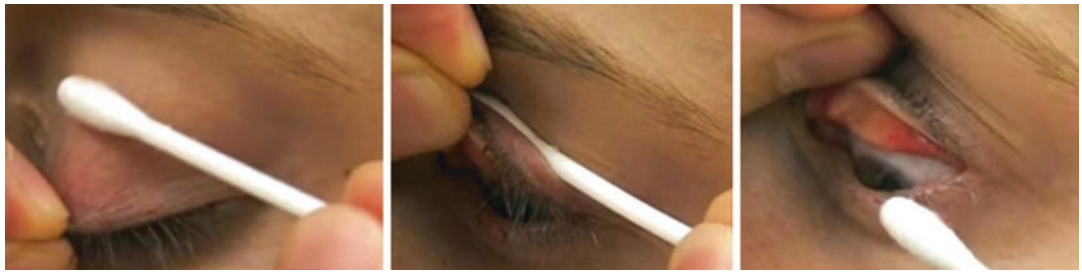

Fig. 3.7 After anesthetizing both eyes, have the patient look downward, then pull downward on the upper eyelashes (left). Place the cotton-tip applicator halfway up from the lid margin gently pushing downward while lifting the lid upward (center). The cotton-tip applicator can now be moistened with sterile saline and used to sweep out any foreign bodies seen under the anterior lid

deeper structures must be considered [28]. This is also the case for an eye that is both red and miotic.

A drop of ophthalmic anesthetic may be therapeutic as well as diagnostic. If ocular pain is relieved by topical anesthetic, the differential diagnosis leans toward superficial pathologies such as conjunctivitis, corneal injuries, and foreign bodies. If the pain is not relieved, it suggests a deeper ocular pathology such as iritis or endophthalmitis [24, 28, 30].

Pupil Exam

Three things must be evaluated: shape, size, and reaction to light [31]. Pupillary reaction is broken down further to equality of response, consensual reflex, and accommodation. Size and reaction of each pupil must be compared to the other. Trauma may complicate pupillary exam. Periorbital edema and blepharospasm (reflexive squeezing of the eyelid to external stimuli) will make eye opening difficult. In this situation, eyelids must be forced to open without applying direct pressure on a potentially ruptured eye. To do so, press on the frontal bone at the superior orbital rim and the maxilla at the inferior orbital rim. This will weaken the muscles that close the lids. Next, retract the lids from the superior and inferior orbital rims to open them without placing pressure on the globe [8, 28]. Use gauze to improve grip on the skin, which may be slippery.

The most common cause of an irregularly shaped or non-reactive pupil is prior ocular surgery, most often for cataracts [26]. Physicians must take this into account and correlate the finding with other aspects of history and exam. In a traumatic setting, a teardrop-shaped pupil may suggest globe rupture. The apex of the teardrop points to the laceration [24, 28, 32]. Another cause of irregular pupil is synechiae, or scarring, from previous iritis.

Pupil size should be measured in millimeters. There are reference tools available to accurately measure the pupil size; however, it is more important to determine pupil size relative to the other. When pupil sizes are different, this is called anisocoria. The most common cause is physiologic and benign [29, 31]. A single dilated pupil may also raise suspicion for a "blown" pupil caused by compression of the third cranial nerve in an unconscious patient. This is especially concerning if an impending uncal herniation is suspected. If the patient is awake and alert, this may be due to incidental contamination of the eye with a mydriatic agent. With physiologic anisocoria, the pupil size should be consistently different and change equally under different lighting. In a dim environment, the pupils should dilate equally. In a bright environment, the pupils should constrict equally. When the smaller pupil does not dilate appropriately under dim light or the larger pupil does not constrict appropriately under bright light, there is a pathologic miosis or mydriasis, respectively [6, 28]. Another way to evaluate for baseline aniso-

coria is to examine the patient's identification card or past photographs. If the picture quality is adequate, the provider may be able to compare pupil size.

Pupillary responses to both direct and consensual light stimulation should be recorded. Normal pupils will constrict equally to direct light. A normal pupil will also display consensual reflex, constricting equally when light is flashed over the other eye. Consensual light reflex is tested by the "swinging flashlight test" which may detect relative afferent pupillary defect (RAPD) suggestive of an optic nerve pathology. This test is performed by swinging the light back and forth from one eye to the other to observe each pupil's reactions. In a normal eye, the pupil will constrict under direct light. When this happens, the consensual reflex will make the other pupil also constrict. In a patient with RAPD, when the light swings over to the abnormal eye, that pupil will then abnormally dilate back to its usual, mid-range size. This is because the abnormal pupil was constricted due to the consensual reflex when the light was directly over the normal eye. In its inability to process the light, the abnormal pupil will fail to react to direct light, creating the appearance of dilating inappropriately. RAPD is a pathology of sensory system distal to the optic chiasm. Its presence will in general pin the diagnosis to the retina or the optic nerve. Pathologies at and proximal to the optic chiasm toward the occipital lobe will not elicit a RAPD on exam [6, 21, 27].

Accommodation is tested by having the patient focus on a finger held at a distance and then moving it closer to the patient's nose. This is usually done in conjunction with the extraocular movement exam. Patients have normal accommodation when pupils constrict as the finger moves closer. In patients with light-near dissociation, their pupils will not react to light but will accommodate. Light-near dissociation can be seen in several pathologies, including Argyll-Robertson (syphilitic) pupils and Adie's pupils [31].

Visual Acuity

The visual acuity (VA) exam, with only a few exceptions, should never be excluded [28, 29, 33, 34]. The only time VA testing could be temporarily deferred is a patient who had chemical exposure to the eye [21, 22]. In that situation, the eye should be copiously irrigated before pursuing further testing. In general, the exam should be done in a "corrected" manner. Provider must score in a consistent, reproducible manner on the scale of Snellen chart measures, counting fingers, perceiving motion, or perceiving light. Most importantly, acuity of each eye must be isolated for comparison. The VA exam is "corrected" when possible to exclude chronic, refractory pathology. If a patient wears glasses or contacts regularly, leave them on for the VA exam. If unable to do so, pinhole testing can be used to correct refractory effects to at least 20/30. Commercial pinhole devices are available, but they can be made easily by any clinical provider. Simply punch a hole in a notecard with an 18-gauge needle and the patient will have a pinhole to look though [30]. Significant VA deficits after pinhole exam cannot be attributed to simple refractive error.

The affected eye should be tested first followed by the unaffected eye. The unused eye should be covered, not closed by the patient, during testing. Snellen far vision chart testing is the preferred method of VA testing. The chart is positioned at a distance of 20 feet from the patient. The visual acuity is recorded as the lowest line the patient can read more than half the letters and the number of incorrectly read letters. For example, if a patient reads five out of seven from the 20/40 line, his visual acuity is 20/40 − 2 [30]. The numerator is the distance in feet from the chart that the patient can read the line. The denominator is the distance in feet from the chart that a person with normal vision can read the same line. Thus, a 20/40 vision means that a patient can read a line at 20 feet when a person with normal vision can read the same line at 40 feet.

If a patient is unable to read the first, largest letter of the chart at the 20/200 line, several options are available. First, the patient can move closer to the chart until he is able to read the first letter. The numerator will change to the distance at which the patient can read the first letter. For example, if the patient is able to read the first letter at 15 feet, patient's acuity is 15/200. The patient may also attempt to count fingers at a distance; document the furthest distance at which this is possible—"count fingers at ___ feet." If the patient is unable to count fingers, evaluate whether the patient can perceive hand motion. If unable, evaluate whether the patient can perceive light. The most accurate way to evaluate light perception is to shine a light from each of the four visual field quadrants and see if the patient can localize where the light is coming from. If the patient can localize the light, record vision as "light perception with projection." If the patient can perceive light but cannot localize, record vision as "light perception without projection" [26, 27, 31].

Alternatively, a near vision chart (Fig. 3.8), read at 14 inches, can be used in situations where Snellen chart testing is not feasible. This is used in a resource-limited setting or with a patient who physically cannot sit up for the Snellen chart. To ensure precision and accuracy, a 14-inch string can be attached to the bottom of the chart to measure the correct distance from a patient's chart to the cheek. There are other alternatives. For illiterate patients or young children, the "E" chart may be an option [28]. For verbal children, the Allen chart, with pictures that a verbal child can identify, can be used. Make sure to have the child name the pictures at a close distance prior to exam, as the child may have creative names for each picture. VA apps are available for the Android and iPhone platforms for tablets and smartphones.

In infants older than 6 months, the provider may evaluate for the ability to fixate and track. A light may be shined 1–3 feet away from the infant and both eyes are tested simultaneously. Central, steady fixation is equivalent to about 20/40 vision, unsteady fixation is about 20/100, and inability to fixate at all reveals an acuity less than20/400 [35]. Lastly, optokinetic reflex can be

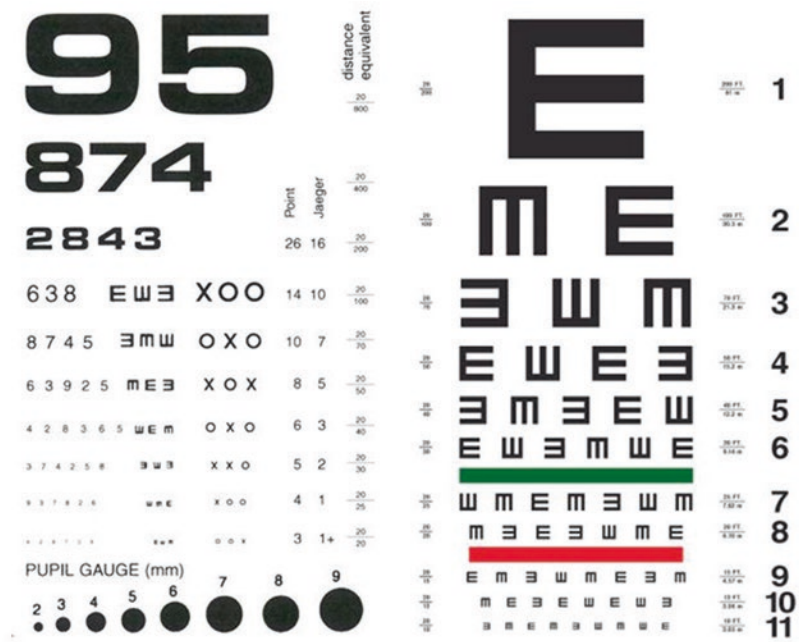

Fig. 3.8 Example of a near vision (14 inch) pocket chart (left) and an "E" Chart (right). For illustrative purpose only (not represented in the proper scale for patient use)

tested using a spinning device (or video on provider's choice of multimedia device) to induce reflexive nystagmus in any patient who can see movement [27, 34]. This test can be used to evaluate vision in newborns or to rule out a physiologic component in patients with feigned total blindness.

Ocular Motility

Extraocular movement (EOM) needs to be carefully evaluated and documented, especially if the patient presents with binocular diplopia (double vision that resolves when covering one eye). Normal eyes should be able to move through the six cardinal directions of gaze without causing double vision [26]. Additionally, the eyes should together converge medially when tracking an object approaching the patient's nose. Test these movements, along with pupillary accommodation, by having the patient follow the provider's finger. The muscle that moves the eye in a direction that causes the greatest separation of the two images is the dysfunctional one. The more peripheral image originates from the pathologic eye [6, 26]. If a patient has normal extraocular function and still complains of double vision, cover one eye and test the other, respectively. If double vision persists in the uncovered eye, this is called monocular diplopia and is suggestive of corneal, lens, or malingering pathology [31].

There are six extraocular muscles that account for the six cardinal directions of gaze: straight nasal (medial rectus), straight temporal (lateral rectus), up nasal (inferior oblique), down nasal (superior oblique), up temporal (superior rectus), down temporal (inferior rectus) (Fig. 3.9).

Three cranial nerves (third, fourth, and sixth) innervate the extraocular muscles. The sixth cranial nerve controls the lateral rectus muscle. The fourth cranial nerve controls the superior oblique muscle. The third cranial nerve controls all other muscles, pupillary constriction, and upper lid elevation. Additionally, medial longitudinal fasciculus (MLF) coordinates left and right third cranial nerves for convergence.

If EOM deficit is noted, consider whether this is due to direct muscle injury, indirect muscle impairment, or neurologic deficit [9]. Extraocular muscle can be indirectly impaired by entrapment from blowout fractures or by retroorbital swelling from orbital cellulitis or hematomas. In setting of facial trauma, entrapment and orbital compartment syndrome must be considered. The combination of significant proptosis, generalized EOM deficit, and acute visual acuity deficit is highly suspicious for orbital compartment syndrome from a retroorbital hematoma. Confirm the diagnosis by measuring the intraocular pressure (see section "Ocular Ultrasound" later) [25]. This is an absolute ophthalmologic emergency requiring immediate ophthalmology consult and lateral canthotomy. In absence of trauma, the presence of periorbital edema, proptosis, EOM deficit, and possibly fever suggests orbital cellulitis. Consider central nervous system (CNS) pathologies such as cranial nerve palsy, CVA, or multiple sclerosis (MS) if the EOM deficits fit a specific nerve pathway. Intranuclear

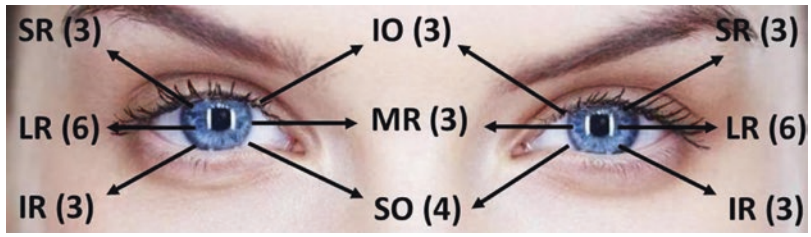

Fig. 3.9 Cardinal directions of gaze of both eyes depicting the extraocular muscles responsible for that direction and the cranial nerve (in parenthesis) that controls that muscle. SR superior rectus, LR lateral rectus, IR inferior rectus, IO inferior oblique, MR medial rectus, SO superior rectus

ophthalmoplegia (INO), an adduction deficit on horizontal gaze usually with preserved convergence, for example, is suggestive of multiple sclerosis (MS) [36]. Pain with any EOM may be due to optic neuritis, also concerning for MS.

Motility testing in children and comatose patients requires the use of other techniques. For young children, providers may utilize different tools to attract the child's gaze toward each cardinal direction. Children are easily bored by the same stimulus. Consider the dictum "one toy, one look" [28]. For neonates, doll's eye maneuver can be performed. Keep the infant calm and make sure eyes are open. Turn the head left or right. The eyes should move in the direction opposite of the turn. Alternatively, this can also be performed by holding the baby facing the examiner and rotating the baby's body left and right [28, 30, 35]. It is important to note that cranial nerve six palsy (lateral rectus deficit) in a child has 50% likelihood of being caused by a neoplasm [9]. Immediate referral is needed in this case. For comatose patients without concern for cervical spine injury, the doll's eye maneuver can also be used. If cervical spine injury cannot be excluded, consider caloric testing [6, 27].

Visual Field Testing

Patients are often unaware of their field deficits or have trouble describing them. Therefore, formal testing is crucial to an accurate diagnosis [6, 31]. Gross confrontational visual field testing can be performed by having the examiner and the patient face each other, with their eyes at the same level. The patient covers one eye, and the examiner covers the opposite eye. The patient looks at the examiner's nose, and the examiner holds his or her fingers half way between him/herself and the patient. The examiner puts his or her fingers in each of the four quadrants, starting far way and moving in progressively toward the nose. The physician may wiggle the fingers while moving inward. The physician should be sure that the patient does not look away from the nose and toward the fingers. Repeat the process in the opposite eye. When the examiner can see the fingers but the patient cannot, a visual field defect is likely and more formal testing is required [20, 26, 27].

If a visual field defect is suspected or detected, the defect should be mapped to localize the pathology. It is important to remember that defects are not always obvious, complicating localization. For example, a partial, monocular field deficit is suggestive of retinal detachment or branch retinal artery occlusion. However, a subtle, incongruous, and incomplete homonymous hemianopia from the other eye may have been missed. That finding would localize the lesion to an incomplete chiasmal defect (Fig. 3.10).

Direct Visualization of the Anterior Chamber

The most important aspect of the anterior chamber exam is to identify corneal defects such as abrasion, keratitis, ulcer, and foreign body. Few pathologies can be seen by direct, unaided visualization. This includes large foreign bodies, keratitis from large corneal ulcers, and corneal clouding from glaucoma or alkaline chemical exposure, for example. Definitive diagnosis of anterior chamber structural pathologies, however, requires magnification using a slit lamp and visualization of the fluorescein-stained eyes under a Wood's lamp or cobalt blue light of a slit lamp (Fig. 3.11).

Ideally, slit lamp exam should be performed first without fluorescein staining. Epithelial defects over the cornea may be noted by the skilled examiner. A small patch of corneal clouding is suggestive of a corneal ulcer. It does not need a stain to visualize. Corneal abrasions, in contrast, may also be visualized without aid of fluorescein; however, they do not in general obscure the cornea. The slit lamp may also identify other findings such as cell and flare in the anterior chamber. While these findings suggest urgent concerns such as uveitis or viral conjunctivitis requiring ophthalmic corticosteroids, they do not generally require emergent intervention.

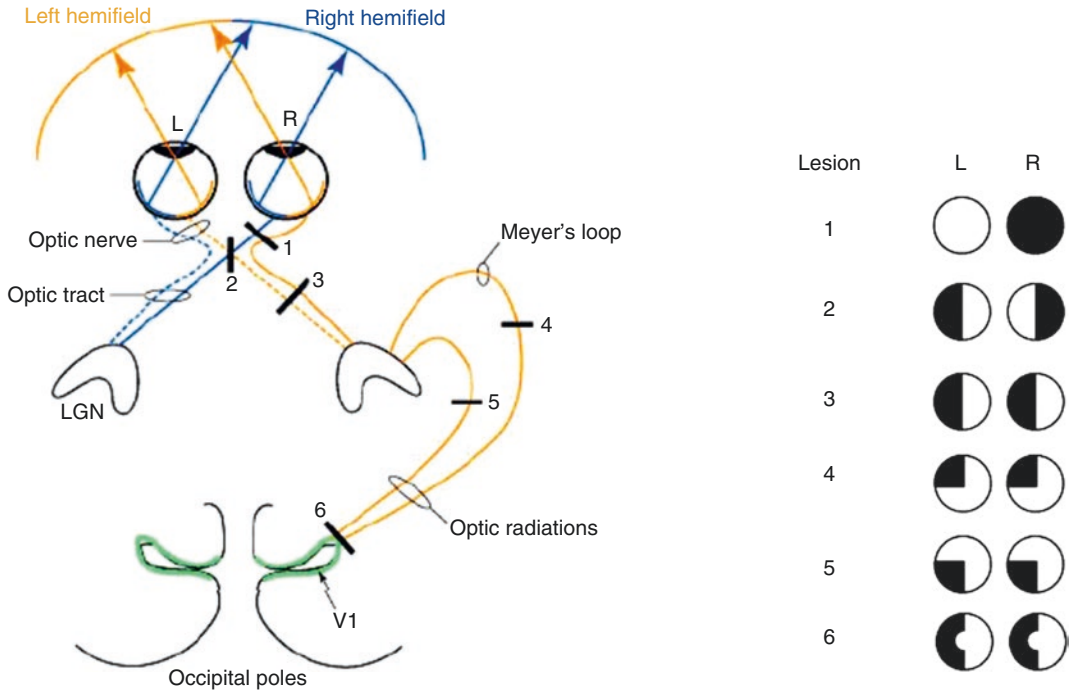

Fig. 3.10 Depiction of the left and right hemifields and the resultant left and right eye visual field deficits (black) from various lesions in the optic neurologic pathways (1). Loss of right eye, (2) loss of peripheral vision due to loss of input from both nasal retinas, (3) loss of left visual field (homonymous hemianopsia), (4) left upper quadranopsia, (5) left lower quadranopsia, (6) cortex lesion with macular sparing

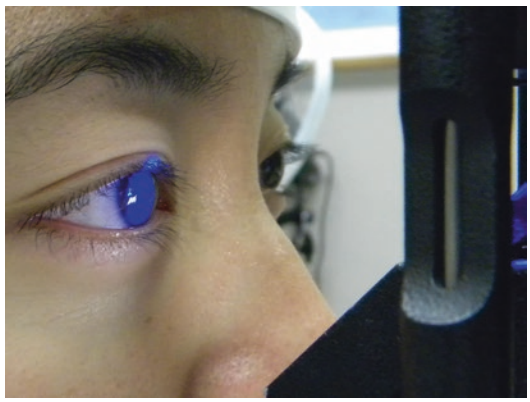

Fig. 3.11 Use of cobalt blue light on the slit lamp after instilling a topical anesthetic agent into both eyes (to blunt the consensual blink reflex), and fluorescein staining of the affected eye will enhance the ability to detect corneal pathology

Intraocular Pressure Measurement

IOP measurement is generally done to diagnose acute angle-closure glaucoma. In traumatic setting, it is performed to diagnose orbital compartment syndrome. It is not necessary in every case. Intraocular pressure measurement is contraindicated in patients with suspicion for open globe, and is relatively contraindicated in patients with active corneal pathologies such as an infection or epithelial defect. While IOP measurement is important, one must balance its value with potential for worsening the existing pathology. The normal IOP is about 12 mm Hg and increases by 1 mm Hg per decade after the age of 40 years. In general, it is considered normal under 20 mm Hg. In patients with acute angle-closure glaucoma, the IOP the is around 50–70 mm Hg. In children, formal measurement is rarely required and if necessary, should be performed by an ophthalmologist.

The techniques for measuring IOP are typically device and patient position dependent (Fig. 3.12). Traditionally, the Shiotz tonometer was used. This device is field and combat durable and does not require batteries. More commonly

Fig. 3.12 Shiotz (left), Tono-pen® (middle), and icare® (right) tonometers

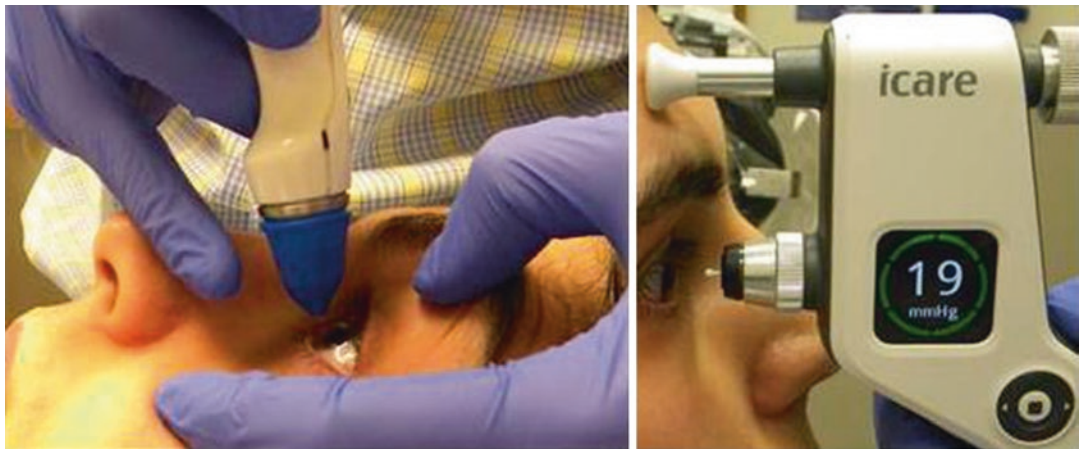

Fig. 3.13 Like the Shiotz tonometer, the Tono-pen® requires the patient to be supine (left), while the icare® is used in patients who can sit upright

used now are several models of electronic battery-powered devices: Tono-pen® tonometers (Reichert Technologies) and icare® tonometers (icare USA). The Tono-pen® requires corneal anesthesia and a disposable latex tip and is used in supine patients. The advantage of the icare® device is that it does not require corneal anesthesia; however, its use is limited to vertical patient positioning (Fig. 3.13).

Visualization of the Posterior Chamber

Traditionally, a full ophthalmologic exam includes direct ophthalmoscopy by the emergency physician to evaluate the posterior chamber. This exam is difficult without chemical dilation of the pupils. Dilation is contraindicated in patients requiring serial neurologic exams, especially in the setting of traumatic brain injury without neurosurgical clearance [29]. It is also contraindicated in patients who have had cataract surgery and patients who have had acute angle-closure glaucoma in the past [37]. Direct ophthalmoscopy using a PanOptic™ Ophthalmoscope (Welch Allyn) is easier to use than a traditional ophthalmoscope (Fig. 3.14). The optics system converges the light to a point at the cornea, which allows the examiner easy entry into small pupils. The illumination pathway then diverges to the retina, illuminating a very wide area of the

Fig. 3.14 PanOptic™ ophthalmoscope (Welch Allyn)

fundus. The viewing system enables the examiner to view the illuminated area on virtually the same axis, thus creating the widest field of view attainable in undilated ophthalmoscopy [38].

During the ophthalmoscopy exam, the following are considered concerning findings [9, 22]:

1. Central retinal artery occlusion (CRAO)—a pale fundus with a cherry red spot at the macula suggestive of retinal ischemia.
2. Retinal vein occlusion (RVO)—venous dilation and diffuse, large hemorrhages throughout the retina.
3. Retinal detachment—retinal surface with appearance of billowing sale or sand dunes at the area of detachment. Progression beyond the macula determines whether the detachment is macula on or off.
4. Vitreous hemorrhage—sometimes the fundoscopic view may be entirely occluded by blood in the posterior chamber.
5. Papilledema—optic disc margin, which is usually sharply demarcated, is blurred, suggestive of elevated intracranial pressure (ICP).

Ocular Ultrasound

While fundoscopy is the definitive method to evaluate the posterior chamber, it may be technically difficult for the non-ophthalmologist. It is made more difficult if the eyes are not dilated or have structural abnormalities such as corneal defect, dense cataract, or vitreous hemorrhage [39]. In these situations, ocular ultrasonography may be easier to perform and yields useful, structural information about a patient's globe, its chambers, and the optic nerve.

In the emergency department, ocular ultrasound is performed with a high-resolution (7.5 MHz or higher) linear array transducer. This will yield images similar to the B-scan obtained in an ophthalmologist's office. To scan, apply a large amount of water-soluble ultrasound gel over the patient's closed eyelid (Fig. 3.15). Place the probe over the gel on the closed eyelid without exerting significant pressure on the eye. Adjust the depth so that the image of the eyeball fills the screen. Adjust the gain to achieve acceptable image. Scan both eyes in sagittal and transverse planes. Ask the patient to look straight ahead with eyes closed, but without clenching the eyelids. While scanning, ask the patient to move his eyeballs as if he is looking up, down, left, and right [40]. For cleanliness, use a probe cover or place a thin, transparent adhesive dressing over the closed eyelid. It is important to avoid trapping air between the skin and the dressing or between the probe and the probe cover. Presence of air between the transducer and the eye will significantly degrade image quality.

Ultrasound can be very useful for detecting retinal detachment and vitreous hemorrhage. If a retinal detachment is present, the examiner will see a highly echogenic (bright), undulating membrane in the posterior chamber (Fig. 3.16).

Initially, the detached retina is highly mobile with eye movement, but with time, the retina becomes stiffer. Echogenic pattern of a vitreous hemorrhage depends on its age and severity. In fresh, mild hemorrhages, there will be small dots or linear areas of mildly echogenic, mobile vitreous opacities. In severe, older hemorrhages, blood will organize and form membranes, filling the usually hypoechoic (dark) posterior chamber with large opacities [41]. In one study, ultrasound in non-traumatic setting had a sensitivity and specificity of 92.3% and 98.3% for retinal detachment, and 100% and 100% for vitreous hemorrhage [42].

Fig. 3.15 Technique for ocular ultrasound

Optic nerve sheath diameter (ONSD) correlates with ICP and can be measured with an ultrasound. First, find the optic nerve by identifying the hypoechoic tract seen perpendicular to the posterior wall of the globe (Fig. 3.16). Where this tract meets the hyperechoic border of the globe is the optic disc. To measure ONSD, take the diameter measurement at 3 mm posterior to the optic disc at high gain. Average two measurements. ONSD greater than 5 mm correlates with ICP greater than 20 mm Hg. This exam has a sensitivity ranging from 88% to 96% and specificity ranging from 76% to 94% for elevated IOP [43–45].

Several traumatic pathologies of the eye may be evaluated with the ultrasound. In globe rupture, the ultrasonographer may note irregularities in shape or size of the globe. The anterior chamber may be collapsed [46]. Lens subluxation or dislocation may be seen. Intraocular foreign body

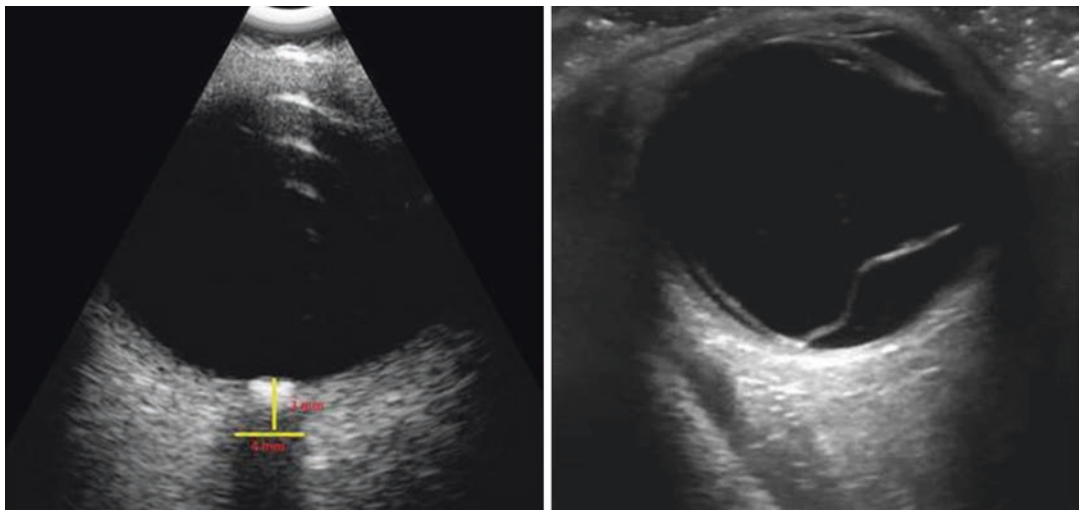

Fig. 3.16 Bedside ultrasound of optic nerve sheath diameter measurement (left) and retinal detachment (right)

will have a bright, echogenic acoustic profile and will often appear with posterior acoustic shadow or reverberation artifact [47].

While the ocular ultrasound adds significant value to the ocular exam, its image quality is highly user-dependent. Almost all published studies on ocular ultrasonography were performed by highly experienced ocular ultrasonographers. Thus, its high sensitivity and specificity will likely be much lower in reality in the emergency setting. Any diagnosis made by the emergency provider's ultrasound scan needs to be confirmed by an ophthalmologist.

References

1. Sharma R, Brunette DD. Chapter 71 – Ophthalmology. In: Marx JA, Hockberger RS, Walls RM, et al., editors. Rosen's emergency medicine concepts and clinical practice. 8th ed. Philadephia: Elseiver Saunders; 2014.
2. Gendler S, Nadler R, Erlich T, et al. Eye injury in the Israeli Defense Force: "an ounce of prevention is worth a pound of cure". Injury Int J Care Injured. 2015;46:1241–4.
3. Ari AB. Eye injuries on the battlefields of Iraq and Afghanistan: public health implications. Optometry. 2006;77:329–39.
4. Weichel ED, Colyer MH. Combat ocular trauma and systemic injury. Curr Opin Ophthalmol. 2008;19:519–25.
5. Thach AB, Johnson AJ, Carroll RB, et al. Sever eye injuries in the war in Iraq 2003–2005. Ophthalmology. 2008;115:377–82.
6. Parr J. Examination. In: Introduction to ophthalmology. Oxford: Oxford University Press; 1989. p. 92–110.
7. Roper-Hall MJ. Symptoms and circumstances causing attendance at A&E departments. Eye emergencies. New York: Churchill Livingstone; 1987. p. 9–17.
8. Clark RB, Farber JM, Sher NA. Eye emergencies and urgencies. Patient Care. 1989;23:24–42.
9. Silverman H, Nunez L, Feller DB. Treatment of common eye emergencies. Am Fam Physician. 1992;45:2279–87.
10. Friedberg MA, Rapuano CJ. Trauma. In: Wills Eye Hospital – office and emergency room diagnosis and treatment of eye disease. Philadelphia: JB Lippincott; 1990. p. 17–45.
11. Andreoli C, Gardiner M. Open globe injuries: emergency evaluation and initial treatment. UpToDate. Assessed 17 Oct 2017.
12. Al-Omaran AM, Abboud EB, Abu El-Asrar AM. Microbiologic spectrum and visual outcomes of posttraumatic endophthalmitis. Retina. 2007;27(2):236.
13. Duch-Samper AM, Chaques-Alepuz V, Menezo JL, Hurtado-Sarrio M. Endophthalmitis following open-globe injuries. Curr Opin Ophthalmol. 1998;9(3):59.
14. Affeldt JC, Flynn HW Jr, Forster RK, Mandelbaum S, Clarkson JG, Jarus GD. Microbial endophthalmitis resulting from ocular trauma. Ophthalmology. 1987;94(4):407.
15. Ansari MH. Blindness after facial fractures: a 19 year retrospective study. J Oral Maxillofac Surg. 2005;63:229–37.
16. Fattahi T, Brewer K, Retana A, Ogledzki M. Incidence of retrobulbar hemorrhage in the emergency department. J Oral Maxillofac Surg. 2014 Dec;72(12):2500–2.
17. Lee KYC, Tow S, Fong KS. Visual recovery following emergent orbital decompression in traumatic retrobulbar haemorrhage. Ann Acad Med Singap. 2006;5(11):831–2.
18. Winterton JV, Patel K, Mizen KD. Review of management options for a retrobulbar hemorrhage. J Oral Maxillofac Surg. 2007;65(2):296–9.
19. Burns FR, Peterson C. Prompt irrigation of chemical eye injuries may avert severe damage. Occup Health Saf. 1989;58:33–6.
20. Garcia GE. Ophthalmology for the non-ophthalmologist: I. Minor office emergencies. Emerg Med. 1987;19:62–7.
21. Shingleton BJ. Eye injuries. NEJM. 1991;325:408–13.
22. Janda AM. Ocular trauma. Postgrad Med. 1991;90:51–60.
23. Arredondo AR, Dire DJ. Assessment of tetanus risk in the pediatric emergency department. Pediatr Emerg Rep. 2016;21(12):157–68.
24. Melamed M. The injured eye at first sight. Emerg Med. 1989;20:86–9.
25. Lima V, Burt B, Leibovitch I, Prabhakaran V, Goldberg RA, Selva D. Orbital compartment syndrome: the ophthalmic surgical emergency. Surv Ophthalmol. 2009;54(4):441–9.
26. Frie JC. Ophthalmic history and examination. In: Bartley GB, Liesegang TJ, editors. Essential of ophthalmology. Philadelphia: JB Lippincott; 1992. p. 3–25.
27. Catalano RA. Examination of the eye. In: Ocular emergencies. Philadelphia: WB Saunders; 1992. p. 3–43.
28. Levin AV. Eye emergencies: acute management in the pediatric ambulatory care setting. Pediatr Emerg Care. 1991;7:367–77.
29. Kut LJ, Moran DD. Examination of the emergency eye patient. In: Wilenski JT, Read JE, editors. Primary ophthalmology. Orlando: Grune & Stratton; 1984. p. 3–17.
30. Sklar DP, Lauth JE, Johnson DR. Topical anesthesia of the eye as a diagnostic test. Ann Emerg Med. 1989;18:1209–11.
31. Handler JA, Ghezzi KT. General ophthalmologic examination. In: Scott JL, Ghezzi KT, editors.

Emergency treatment of the eye. Emerg Med Clin North Am. 1995;13(3):521–538.
32. Deutsch TA. Ocular emergencies in childhood. Pediatrician. 1990;17:173–6.
33. Clancy MJ, Hulbert M. A study of the eye care provided by an accident and emergency department. Arch Emerg Med. 1991;8:122–4.
34. Gregory-Roberts J. Pitfalls in penetrating eye injuries. Med J Aust. 1992;157:398–9.
35. Read JE. Ocular examination of the neonate and small infant. In: Wilenski JT, Read JE, editors. Primary ophthalmology. Orlando: Grune & Stratton; 1984. p. 19–26.
36. Muri RM, Meienberg O. The clinical spectrum of internuclear ophthalmoplegia in multiple sclerosis. Arch Neurol. 1985;42(9):851–5.
37. Luff A, Elkington A. Better use of ophthalmoscope. Practitioner. 1992;236:162–5.
38. A guide to the use of diagnostic instruments in eye and ear examinations. Skaneateles Falls: Welch Allyn; 2006.
39. Qureshi MA, Laghari K. Role of B-scan ultrasonography in pre-operative cataract patients. Int J Health Sci. 2010;4(1):31–7.
40. Babineau MR, Sanches LD. Ophthalmologic procedures in the emergency department. Emerg Med Clin North Am. 2008;26(1):17–34.
41. Byrne SF, Green RL. Ultrasound of the eye and orbit. 2nd ed. St. Louis: Mosby Year Book; 2002.
42. Parchand S, Singh R, Bhalekar S. Reliability of ocular ultrasonography findings for pre-surgical evaluation in various vitreo-retinal disorders. Semin Ophthalmol. 2014;29(4):236–41.
43. Kimberly HH, Shah S, Marill K, Noble V. Correlation of optic nerve sheath diameter with direct measurement of intracranial pressure. Acad Emerg Med. 2008 Feb;15(2):201–4.
44. Moretti R, Pizzi B. Optic nerve ultrasound for detection of intracranial hypertension in intracranial hemorrhage patients: confirmation of previous findings in a different patient population. J Neurosurg Anesthesiol. 2009;21(1):16–20.
45. Rajajee V, Vanaman M, Fletcher JJ, Jacobs TL. Optic nerve ultrasound for the detection of raised intracranial pressure. Neurocrit Care. 2011;15(3):506–15.
46. Blaivas M, Theodoro D, Sierzenski P. A study of bedside ocular ultrasonography in the emergency department. Acad Emerg Med. 2002;9:791–9.
47. Shriver SA, Lyon M, Blaivas M. Detection of metallic ocular foreign bodies with handheld sonography in a porcine model. J Ultrasound Med. 2005;24:1341–6.

Damage Control Ophthalmology: Anesthesia Considerations

4

Colonel Mark H. Chandler

Abbreviations

ABC	Airway breathing circulation
ASA	American Society of Anesthesiologists
BETTS	Birmingham Eye Trauma Terminology System
cc	cubic centimeter
CN	Cranial nerve
ETT	Endotracheal tube
FDP	Face-down position
GETA	General endotracheal anesthesia
IED	Improvised explosive device
IOP	Intraocular pressure
JTTR	Joint Theater Trauma Registry
LA	Local anesthesia
MARCH	Massive hemorrhage, airway, respiration, circulation, head/hypothermia
mL	milliliter
NDMR	Nondepolarizing muscle relaxant
NPO	nil per os (nothing by mouth)
OCR	Oculocardiac reflex
PBB	Peribulbar block
RBB	Retrobulbar block
USA	United States of America

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Introduction

Despite improvements in and the widespread use of protective tactical eyewear, eye trauma remains an important problem for military surgeons and anesthesiologists on the modern battlefield. Indeed in the recent conflicts in Iraq and Afghanistan, the increased use of explosive weapons such as improvised explosive devices (IEDs), coupled with the extensive use of body armor ensuring soldiers survive mechanisms that would have proved mortal in previous conflicts, has led to a paradoxical increase in eye injuries. A study using data from the Joint Theater Trauma Registry (JTTR) indicates that from October 2001 to January 2005 (still early in the conflicts in Iraq and Afghanistan), eye wounds accounted for 380, or 6% of 6609 wounds sustained by 3102 casualties [1]. Future conflicts will likely prove just as costly to our soldiers' vision, and for this the astute military medical provider must be well prepared.

Anatomy and Physiology

The complex structure and functioning of the eye is covered in greater detail elsewhere in this volume; however, a rudimentary understanding of ocular anatomy and physiology is presented here as it relates to anesthesia of the eye.

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The sclera is a fibrous protective outer sheath that gives the eye its spherical shape and forms the "white" of the eye. The pigmented iris is a thin, epithelial and fibrous circular structure that controls the diameter and size of the pupil, and also divides the anterior chamber (covered by the transparent cornea) from the posterior chamber of the eye. Near the outer edge of the iris is the ciliary body, which is comprised of both the ciliary muscle, which controls the shape of the lens, and the ciliary epithelium which produces aqueous humor. The retina is the light-sensitive posterior portion of the eyeball that sends nerve impulses to the brain via the optic nerve.

Blood is supplied to the eye by the ophthalmic artery, which, after leaving the internal carotid artery, divides into the retinal and uveal circulations. The retinal circulation is supplied by the central retinal artery, which travels in or with the optic nerve as it passes into the sclera to branch and supply the retina. Uveal circulation is provided by the short and long posterior ciliary arteries, and the anterior ciliary artery. Venous drainage of the eye begins with the vortex veins and central retinal vein, which help to form the superior and inferior ophthalmic veins, eventually ending in the pterygoid venous plexus, the cavernous sinus and the facial vein.

The motor and sensory innervation of the eye and surrounding structures is complex. Motor innervation of the eyelid is via the facial nerve (CNVII), the oculomotor nerve (CNIII), and sympathetic nerve fibers. Eye movement is controlled principally by the oculomotor nerve (CNIII), as well as the trochlear nerve (CNIV) and the abducens nerve (CNVI). Sensory innervation of the eyeball itself is supplied by the nasociliary branch of the ophthalmic nerve (V1, first branch of the trigeminal nerve, CNV), which also gives rise to the infratrochlear nerve, which provides sensation to the eyelids, conjunctiva, and lacrimal sac. The ophthalmic nerve also gives rise to the lacrimal nerve, which runs to the lacrimal gland, the conjunctiva, and the skin of the superior eyelid; as well as the frontal nerve, which provides sensation to the eyelid, scalp, and forehead. The ciliary ganglion contains postsynaptic parasympathetic nerve cell bodies and gives rise to the short ciliary nerves, which innervate the ciliary and sphincter pupillae muscles and carry afferent sensory information from the iris and cornea. The long ciliary nerves (usually two to three in number) bypass the ciliary ganglion and provide sensation to the eyeball, and carry sympathetic information to the dilator pupillae muscle.

This complex system of nerves to the eye and surrounding structures make blocking it very challenging and nuanced. To achieve the standard components of regional anesthesia, namely amnesia, analgesia, and muscle relaxation, one must block branches of five separate cranial nerves (CNs II, III, IV, V, VI). Additionally, one needs to block branches of CNVII to prevent the movement of periocular muscles and forceful eyelid closure. Fortunately, most of these nerves are in relatively close proximity to one another, and thus blocking them can be achieved with one or two well and carefully placed injections of local anesthesia.

Intraocular Pressure

Maintenance of intraocular pressure (IOP) within the normal range of 10–20 mm Hg is achieved through a careful balance of aqueous humor production and drainage. Nearly two-thirds of aqueous humor is produced by active secretion of the ciliary bodies, which are regulated by the carbonic anhydrase and cytochrome oxidase systems. Passive filtration in the anterior chamber of the iris accounts for the remaining aqueous humor production. Aqueous humor flows anteriorly through the pupil where it is absorbed by a trabecular network at the angle formed by the iris and the cornea, eventually making its way to the venous system via the canal of Schlemm. Intraocular pressures greater than 22 mm Hg are considered pathologic and if prolonged, can lead to permanent visual loss. Temporary minor elevations in IOP occur with normal physiologic phenomenon such as lying supine and coughing; much greater elevations occur with retching and vomiting, but are usually well tolerated if short in duration. Traumatic mechanisms such as external

compression or hemorrhage within the eyeball can lead to dangerous sustained increases in IOP.

The Birmingham Eye Trauma Terminology System (BETTS)

A team of researchers from the University of Alabama developed the Birmingham Eye Trauma Terminology System (BETTS) in response to the lack of standardized terminology for eye injuries. First described in 1996 [2] but formally named and published in 2002 [3], BETTS has since become the standard for eye injury classification (Fig. 4.1).

This classification system is discussed in greater detail elsewhere in this volume; however, for the purposes of understanding anesthesia of the traumatized eye, note that an "open globe" injury is a full-thickness wound through the sclera and cornea, and thus wounds that fail to penetrate both of these structures are considered "closed globe" injuries [3].

Preoperative Assessment

On the modern battlefield, eye injuries often occur in conjunction with other traumatic injuries. Naturally, life-threatening injuries must be first addressed using time-honored prioritization systems such as "ABC" (airway, breathing, and circulation) or, more appropriate for the battlefield, MARCH (massive hemorrhage, airway, respiration, circulation, and head/hypothermia). Once stabilized, it is important to identify the extent of ocular injury, particularly whether the globe is open or closed, and whether there is other midfacial trauma such as a cribriform plate fracture, which may preclude safe nasotracheal intubation or the placement of a nasogastric tube. Other injuries often found in association with ocular trauma are nasal, mandibular, and maxillary fractures, which may make mask ventilation and placement of an endotracheal tube (ETT) challenging.

Although most victims of combat trauma are young and healthy, it is important, as time allows and the situation dictates, to query any patient undergoing anesthesia about pre-existing disease, prior anesthetics and complications, current medications, and drug allergies. NPO status is a standard part of the preanesthetic rubric, although in the setting of trauma all patients are considered "full-stomachs."

Minimizing the risk of aspiration of gastric contents remains a debated subject in the setting of trauma. Placement of oral or nasal tubes should be avoided when an open globe is known or suspected as this can lead to retching and vomiting,

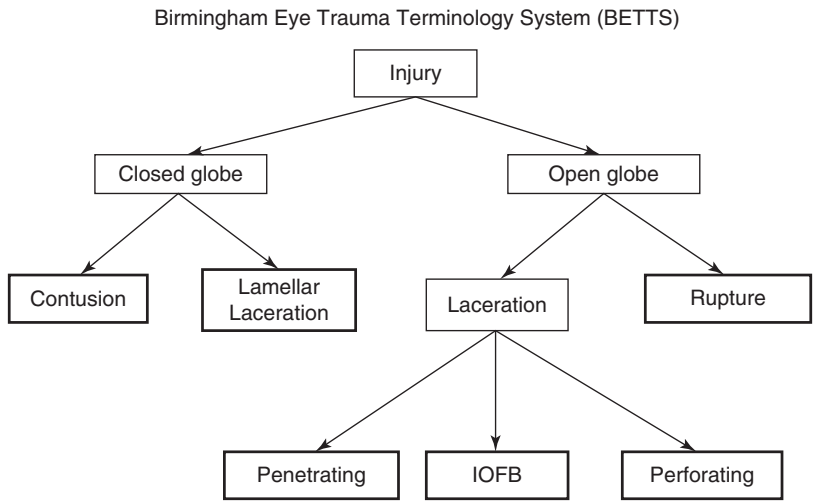

Fig. 4.1 Birmingham Eye Trauma Terminology System (BETTS). (Courtesy of Ferenc Kuhn)

which may cause the expulsion of ocular contents. Generally speaking, most pharmacologic interventions commonly used to prevent or diminish the impact of aspiration are either impractical, not available, or require more time to work than what is typically available in the setting of combat trauma. However, the following agents are often used in elective surgeries:

A. Clear, nonparticulate oral antacids: 30 ml of citric acid/sodium citrate taken immediately prior to induction increases the pH of gastric contents at the cost of slightly increasing gastric volume [4].
B. Histamine-2 receptor agonists (H2RA): Famotidine 20 mg IV or ranitidine 50 mg IV increases pH and reduces the volume of gastric contents, but requires at least 40 minutes to act [5].
C. Prokinetic gastrointestinal agents: With an onset of action in 1–3 minutes, 10 mg IV metoclopramide (Reglan) will increase lower esophageal sphincter tone and the upper GI tract's response to acetylcholine, thus enhancing motility and accelerating gastric emptying without stimulating secretions. Metoclopramide can also act as a postoperative antiemetic, although it is not very effective at preventing postoperative nausea and vomiting at this dose given at the onset of anesthesia [6].

Local Anesthesia Overview

Local anesthetics (LAs) reversibly block pain by inhibiting sodium influx through voltage-gated sodium channels in the neuronal cell membrane, thus preventing the generation and conduction of action potentials in neurons. All local anesthetics are weak bases, usually prepared as water-soluble hydrochloride salts, and are classified as either "amides" or "esters" based on the linkage between amine and aromatic ring ends. While adverse reactions to local anesthetics (especially esters) are common, true allergies are rare and are usually due to the ester metabolite para-aminobenzoic acid (often referred to as "PABA," an active ingredient in many older sunscreens). Fortunately, this ester sensitivity does not result in cross-reactivity to amides.

Ester local anesthetics include benzocaine, chloroprocaine, and tetracaine; amide local anesthetics include mepivacaine, prilocaine, bupivacaine, and lidocaine (a generally reliable memory tool is that those local anesthetics with two "i's" are amides), the latter two are the most commonly used local anesthetics for ocular procedures.

Topical Anesthesia

Needleless topical application of LA to the eye allows more thorough examination, provides comfort, and offers suitable analgesia for minor procedures such as lacrimal probing and irrigation, examination of nasal structures, and forced duction testing. Indeed, the speed, simplicity, and avoidance of hazardous needle techniques has led to the use of topical local anesthesia for 50% of all cataract phacoemulsifications worldwide [7]. However, topical LA does not produce akinesia or intraocular pressure control and may suppress the normal blink reflex and delay re-epithelialization in instances of corneal abrasion [8]. Topical LA must only be used for uncomplicated procedures (e.g., exam under anesthesia) by experienced surgeons with cooperative patients. There are several topical local anesthetic mixtures suitable for ophthalmic anesthesia, to include tetracaine 0.5% or 1% and proparacaine 0.5%.

Retrobulbar Nerve Block

The "retrobulbar block" (RBB), first described by Knapp in 1884 [9], has historically been the "gold standard" of ophthalmic anesthesia as it affords complete anesthesia of the eye. In the classic method described by Atkinson in 1936 [10], the patient was asked to look "up and inward" as the needle was advanced; however, subsequent research has demonstrated that this in fact puts important posterior optic structures

closer to the path of the needle. The patient is now is asked to look in the exact opposite direction, that is "down and outward" [11], or straight, to safeguard these structures. The RBB is typically performed with a 23G needle placed at the junction of the lateral one-third and medial two-thirds orbital margin. The needle is directed superiorly and medially to enter the Tenon's capsule between the lateral and inferior rectus muscles to a depth of 25–35 mm. The syringe is aspirated once the needle has reached the retro-orbital space and 2–4 cc of local anesthetic is injected. As the RBB does not prevent blinking, it is often accompanied by a van Lint block of the orbicularis oculi muscle (a branch of the facial nerve).

The advantages of the RBB are that it requires little local anesthetic, it sets up very quickly with little chance of an incomplete block thus avoiding repeat injections, and does not cause ecchymosis of the eyelids. Its disadvantages are that it is painful, it requires a supplementary facial nerve block, and it can increase intraocular pressure. But most concerning are the sight- and life-threatening risks involved with placing a needle in such close proximity to the important optic structures. These risks include ocular perforation, retrobulbar hemorrhage, profound oculocardiac reflex, damage to the optic nerve [12], and even brainstem anesthesia from inadvertent injection into the dura, causing confusion, dyspnea/apnea, seizures, and death.

Peribulbar Nerve Block

Because of the risks from retrobulbar injection, the peribulbar block (PBB), which injects LA extraconally rather than intaconally, has largely supplanted the RBB as the primary ocular block. While several injection methods have been described, the PBB is classically delivered with two injections: the first is identical to the single injection of the RBB, only with a shorter needle and with less upward and inward angle; the second is superior and nasal between the medial third and lateral two-thirds of the orbital roof edge. The PBB remains a favorite block among ophthalmologists who have offered the following pointers over the years:

- Use at least 8–15 cc of local anesthetic (much more than that for RBB) to allow spread into the intraconal space and the whole corpus adiposum of the orbit. If enough volume is used, LA will spread superiorly and anteriorly, obviating the need for a second injection or a separate lid block. Note that when this large volume is used, the anatomic distortion that takes place can put anatomical structures at greater risk of damage from a second injection, thus the second injection should only be undertaken if anesthesia is insufficient [7].
- Use a fine needle (25G) to reduce pain on insertion, and consider a short-beveled needle to enhance tactile perception to avoid direct intraneural injection [7].
- Limit needle depth to 25 mm to avoid perforating the optic nerve or the apex of the orbit, or injection through the optic foramen [7].
- While compression over the closed eye has not been shown to improve the quality of the block, it has been shown to diminish the increase in intraocular pressure that often follows the PBB [13].
- If a second injection is necessary, avoid placing it too medially in the superior nasal region [7].

Sub-Tenon Nerve Block

Also known as episcleral block or parabulbar, this block places local anesthesia into Tenon's capsule, which is the socket within which the eye moves, formed by a thin layer of connective tissue that surrounds the globe from the optic nerve to the limbus. Two techniques are used to deliver the sub-Tenon block:

- Needle method: a needle is introduced into the fornix between the semilunaris fold of the conjunctiva and the globe, angled tangentially to the globe. After initial penetration, while the patient is gazing medially, the needle is shifted medially and advanced

posteriorly until a small click or loss of resistance is encountered at about 10–15 mm. The patient then returns to the primary gaze position (forward) as up to 10 cc of local anesthesia (depending on the patient's size) is injected [7].

- No-needle or surgical method: generally preferred as it avoids introducing a sharp needle to the optic structures, the no-needle sub-Tenon block is performed after the conjunctiva is anesthetized with topical local anesthesia. Many practitioners favor placing an eyelid speculum as the patient is asked to gaze medially and inferiorly. The conjunctiva is then delicately raised using fine non-toothed forceps midway between the limbus and the visible edge of the inferonasal portion of the conjunctiva. A 2–3-mm opening is created in the conjunctiva and Tenon's capsule followed by either a specialized blunt catheter or a shortened 18- or 20-G plastic intravenous catheter to allow a 3–5-mL injection of local anesthetic. The sub-Tenon block typically does not raise intraocular pressure significantly, and therefore post-injection compression is not necessary. The sub-Tenon block provides excellent global anesthesia but does not usually provide complete akinesia of the globe and lid, thus supplemental anesthesia is often required. The ease and speed with which it can be delivered, however, make it the ideal intraoperative supplemental or "rescue" block when additional anesthesia is necessary [7].

General Anesthesia

On the modern battlefield, most instances of significant eye trauma are accompanied by other often very serious injuries, thus making general endotracheal anesthesia (GETA) the preferred method of creating optimal operating conditions. In most instances, a GETA can be safely administered to eye trauma patients, but the experienced anesthesiologist must anticipate and plan for many potential challenges.

Premedication

With an awake and (understandably) anxious eye trauma patient, premedication with a benzodiazepine and narcotic is appropriate and should have no significant effect on IOP. If other associated facial trauma requires a fiberoptic intubation, antisialogogues such as atropine and glycopyrrolate may be administered intravenously with little to no effect on IOP (administered topically, these agents can raise IOP through mydriasis).

Induction Agents

The quest for the ideal induction agent in the setting of trauma, particularly when associated with eye trauma, remains elusive. Etomidate, the long-favored trauma induction agent due to its stable hemodynamic effect, has been shown to actually decrease intraocular pressure; however, it continues to lose ground among trauma anesthesiologists due an ongoing debate regarding its long-term effects on critically ill patients [14–16]. And while much of the trauma world has embraced ketamine over etomidate for a similarly stable hemodynamic profile and the added benefit of profound analgesia, its use in ophthalmic trauma remains controversial due to a nystagmus effect and a debatable tendency to increase intraocular pressure [17–19]. Several older studies have shown that propofol, if not actually lowering IOP, at least diminishes the increase in IOP associated with intubation [20, 21]; however, its profound effect on cardiac output makes it less than ideal as a trauma induction agent. Thus, in the setting of eye trauma and possible hypovolemia from other associated injuries, the anesthesiologist must balance the well-known hemodynamic effect of propofol with its ability to lower intraocular pressure.

Muscle Relaxation

The use of succinylcholine as a muscle relaxant remains controversial among trauma anesthesiologists and ophthalmologists. Introduced in

1952, succinylcholine was embraced by anesthesiologists as a nearly ideal muscle relaxant due to its fast onset, fast offset, and the ideal intubating conditions it affords. However, almost immediately after its introduction the controversy began with two articles from the late 1950s suggesting succinylcholine's role in increasing IOP and the theoretical risk of sight-threatening vitreous extrusion [22, 23]. Despite the fact that these articles were studies of intraocular physiology and only referenced anecdotal accounts of succinylcholine raising IOP, they are often misrepresented as actual case reports and remain stubborn fixtures in the literature surrounding the IOP–succinylcholine debate.

While no definitive studies or case reports over the last 65 years have described vitreous extrusion with succinylcholine, research has shown that it does indeed slightly raise IOP. Various studies demonstrate an intubating dose of succinylcholine increasing IOP by approximately 5–10 mm Hg [24] for less than 10 minutes (Cook). While definitely in the pathologic range, this increase needs to be understood in the context of other physiologic increases in IOP: blinking increases IOP by 5 mm Hg, laryngoscopy by 10–15 mm Hg, coughing by 40 mm Hg, and forceful eyelid closure by up to 70 mm Hg [25–29]. The increase in IOP caused by forceful masking, i.e., the type that may be necessary while awaiting ideal intubating conditions afforded by nondepolarizing muscle relaxants (NDMRs), is unclear. Various mechanisms have been offered over the years to explain the increased IOP seen with succinylcholine, to include simultaneous contraction of extraocular muscles creating circumferential traction on the eyeball, diminished venous flow from the head caused by simultaneous tightening of the neck muscles, and finally choroidal vascular dilation combined with decreased vitreous drainage through the canal of Schlemm.

In more recent years, several studies have cast serious, well-reasoned doubt upon the importance of succinylcholine-induced IOP increase. Among the most convincing and often cited, Libonati et al. performed a retrospective study of 73 open globe surgeries, all induced with succinylcholine, at the Wills Eye Hospital in Philadelphia, PA. After reporting no incidents of vitreous extrusion in this retrospective review, Libonati et al. go on to extrapolate this to a 10-year history at Wills Eye Hospital, with an average of 250 open globe surgeries each year induced with succinylcholine, with no reported instances of vitreous extrusion [30]. Vanchon et al. went as far as to historically research exactly how succinylcholine's association with increased IOP became so ingrained in the anesthesia and ophthalmic literature. Their article notes the frequent specious citing of the Dillon and Lincoff publications from the 1950s (the Dillon article alone was cited 128 times in a 44-year period), and the continued reliance on anecdotal evidence rather than formal case reports [31]. Indeed, in a culture that embraces "primum non nocere," the succinylcholine–IOP debate is illustrative of how difficult it is to dislodge a long-held belief despite a singular lack of scientific evidence.

Going forward, the decision to use succinylcholine in the setting of trauma must be weighed against the (largely theoretical) risks. Factors to consider are the risk of vomiting (which is believed to raise IOP), the difficulty of the airway, the difficulty in masking, and whether the eye is salvageable. An NDMR such as rocuronium may seem like a simple solution; however, NMDRs usually require masking while awaiting optimal intubating conditions (Rocuronium, using an RSI dose of 0.6–1.2 mg/kg, will provide good intubating conditions in less than 60–90 seconds; Rocuronium package insert). Even more importantly, if one is unable to secure the airway, the much longer offset time for NDMRs (Rocuronium at the RSI dose will provide up to 67 minutes of muscle relaxation) may require a prolonged period of forceful masking, which almost certainly raises IOP significantly, while awaiting the return of spontaneous ventilation. Figure 4.2 illustrates a proposed decision-making algorithm for the choice of muscle relaxant in the face of a traumatic open globe. It should be noted that with the introduction and continuing widespread use of sugammadex, the first ever selective relaxant binding agent which can be used to reverse NDMR like rocuronium in about

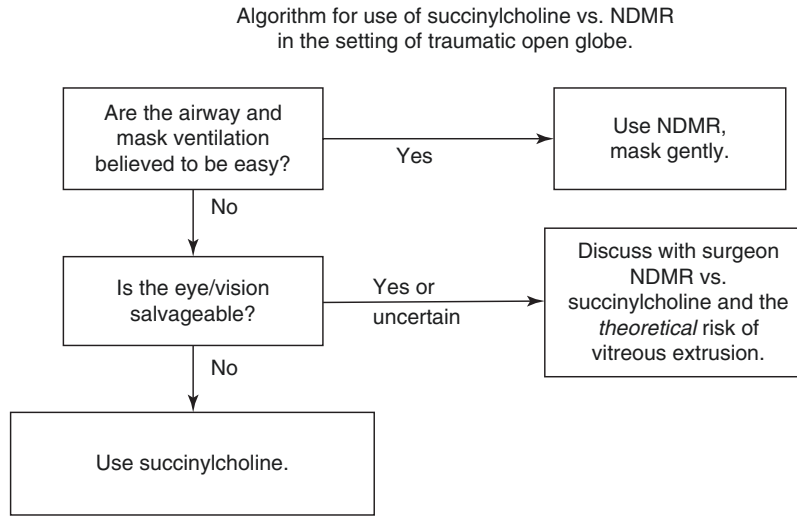

Fig. 4.2 Algorithm for the use of succinylcholine versus NDMR in the setting of traumatic open globe

3 minutes [4], the debate around succinylcholine versus rocuronium will become largely moot [32]. Sugammadex was approved for use in the European Union on July 29, 2008, and for use in the USA by the US Food and Drug Administration on December 15, 2015 [33, 34].

Oculocardiac Reflex (OCR)

Also known as the Aschner phenomenon or the Aschner–Dagnini reflex, the oculocardiac reflex was discovered by Italian researcher Giuseppe Dagnini (1866–1928) and first described by the Austrian physician Berhard Aschner (1883–1960) in 1908. The afferent limb of the reflex comprises the long and short ciliary nerves via the ciliary ganglion to the ophthalmic branch of the trigeminal nerve, which synapse with the visceral motor nucleus of the vagus nerve in the reticular formation of the brain stem. The efferent limb is comprised of the vagus nerve, which transmits information from the cardiovascular center of the medulla to the heart, causing decreased output of the sinoatrial node and bradycardia. Triggers include any ocular manipulation but especially compression of the eyeball or traction of the extraocular muscles (such as during strabismus surgery), particularly the medial rectus muscle. It is important to note that the OCR can be triggered by retrobulbar block itself, by ocular pain, and even with manipulation of the ocular tissue at the apex of the ocular cone following enucleation. The reflex is particularly pronounced in neonates and infants and diminishes with age, but can still be pronounced in adults, particularly young healthy adults with robust autonomic nervous systems (e.g., soldiers). Risk factors in addition to young age include physiologic perturbations such as hypoxia, hypercarbia, and acidosis, and light anesthesia. When profound, the OCR can lead to junctional rhythms, ectopic beats, atrioventricular block, ventricular tachycardia, and asystole. Immediate treatment involves stopping the stimulus and optimizing oxygenation and ventilation. If bradycardia persists, administer atropine 20 mcg/kg or glycopyrrolate 100–200 mcg, recognizing that the slowed heart will significantly decrease circulation time. The best way to prevent severe OCR is with a deep, reliable anesthetic and ongoing communication between the surgical and anesthetic teams.

Monitoring and Maintenance

The monitoring and maintenance of anesthesia for eye trauma cases compared to induction and emergence are very straightforward. No specific monitoring is required for an eye trauma case apart from the standard ASA monitors; additional monitoring, such as an arterial line, CVP,

pulmonary artery catheter, intracranial pressures, etc., may well be prudent in the setting of other associated injuries. Inhalation agents are well tolerated for eye trauma cases and actually decrease IOP through an unknown mechanism [35–39].

In cases of retinal detachment, a gas bubble of sulfa hexafluoride (SF6) or octafluoropropane (C3F8) is injected intraocularly to tamponade the displaced retina. Nitrous oxide, with its high solubility coefficient, can interfere with the repair by "competing" with the gas bubble, thus either causing an undesirable increase in IOP intraoperatively or creating what appears to be an adequate tamponade, only to "deflate" the eye postoperatively by rapidly diffusing out of the vitreous cavity. For these reasons, nitrous oxide should be avoided in retinal detachment repairs.

Emergence

Preventing an increase in IOP during emergence from general anesthesia with a recently traumatized eye can be challenging. Several strategies to prevent coughing and "bucking on the tube" have been proposed over the years. A full reversal of neuromuscular blockade is obviously desirable, and the standard reversal agents, neostigmine and atropine or glycopyrrolate, may be administered with no concern about raising IOP. A "deep emergence," i.e., removing the endotracheal tube while the patient is spontaneously breathing in stage III surgical anesthesia, eliminates gagging on the endotracheal tube, but is less than optimal as it requires a trauma patient with a presumed full stomach to proceed through stage II anesthesia with an unprotected airway. There are, however, many ways to diminish the stimulus of an in-place ETT on emergence, to include administering IV lidocaine (1.5 mg/kg IV), pretreating the ETT with various lidocaine jells or pastes (which can wear off before emergence in long cases), or even using specially designed ETTs that allow the administration of lidocaine beneath the vocal cords prior to awakening (LITA™ cuffed tracheal tubes, CNC Medical Devices).

Physiologic perturbations such as hypoxia, hypercarbia, and acidosis can increase IOP and thus must be avoided during maintenance of anesthesia and upon emergence. Retching or vomiting are also obviously undesirable, and thus prophylactic antiemetics, such as ondansetron 4 mg IV (0.1 mg/kg pediatric dose for ages 1 month to 12 years) and dexamethasone 8 mg IV [37], are often helpful. The recent focus in trauma circles on preventing hypothermia through forced air warming blankets, warm ORs, and fluid warmers is especially important in the traumatized eye patient as postoperative shivering may raise IOP. If these strategies fail, meperidine 25 mg IV is the most commonly used antishivering agent; however, other agents such as clonidine 150 mcg IV and doxapram 100 mg also have proven efficacy [38]. Bandaging the injured eye is usually performed by the surgeon and with an eye patch, although more extensive bandaging is obviously required if there are other facial wounds, in which case care must be taken to prevent pressure upon the injured eye. Finally, the surgeon must be consulted regarding postoperative patient positioning as face-down positioning (FDP) is the standard for certain surgeries (e.g., vitrectomy and gas tamponade procedures), whereas "head-up" positioning is usually preferred for post-open-globe surgeries to prevent increases in IOP.

Conclusion

The modern battlefield offers ample opportunities for eye injuries, as the conflicts in Iraq and Afghanistan so tragically illustrate. While topical anesthetics and ocular blocks have their place for minor procedures, the latter require experience and a practiced hand to administer successfully and avoid complications. The standard of care for significant eye trauma, particularly in the setting of multiple trauma, remains general endotracheal tube anesthesia (GETA). The trauma anesthesiologist must be well versed in the advantages, strategies, and pitfalls of general anesthesia in the traumatized eye patient to preserve the life and vision of the fighting force.

References

1. Owens BD, Kragh JF, Wenke JC, Macaitis J, Wade CE, Holcomb JB. Combat wounds in operation Iraqi Freedom and operation Enduring Freedom. J Trauma. 2008;64(2):295–9.
2. Kuhn F, Morris R, Witherspoon CD, Heimann K, Jeffers JB, Treister G. A standardized classification of ocular trauma. Ophthalmology. 1996;103(2):240–3.
3. Kuhn F, Morris R, Witherspoon CD. Birmingham Eye Trauma Terminology (BETT): terminology and classification of mechanical eye injuries. Ophthalmol Clin N Am. 2002;15(2):139–43.
4. Viegas OJ, Ravindran RS, Shumacker CA. Gastric fluid pH in patients receiving sodium citrate. Anesth Analg. 1981;60(7):521–3.
5. Gan TJ, Meyer TA, Apfel CC, et al. Society for Ambulatory Anesthesia. Society for Ambulatory Anesthesia guidelines for the management of postoperative nausea and vomiting. Anesth Analg. 2007;105(6):1615–28.
6. Henzi I, Walder B, Tramèr MR. Metoclopramide in the prevention of postoperative nausea and vomiting: a quantitative systematic review of randomized, placebo-controlled studies. Br J Anaesth. 1999;83(5):761–71.
7. New York School of Regional Anesthesia [Internet]. Ripart J, Mehrige K, Rocca RD. Local & Regional Anesthesia for Eye Surgery [cited 2018 Jan 10]. Available from: https://www.nysora.com/local-regional-anesthesia-for-eye-surgery.
8. Wang L, Shankarappa SA, Tong R, et al. Topical drug formulations for prolonged corneal anesthesia. Cornea. 2013;32(7):1040–5.
9. Atkinson WS. Retrobulbar injection of anesthetic within the muscular cone. Arch Ophthalmol. 1936;16:494–503.
10. Knapp H. On cocaine and its use in ophthalmic and general surgery. Arch Ophthalmol. 1884;13:402.
11. Liu C, Youl B, Moseley I. Magnetic resonance imaging of the optic nerve in extremes of gaze. Implications for the positioning of the globe for retrobulbar anaesthesia. Br J Ophthalmol. 1992;76(12):728–33.
12. Katsev D, Drews RC, Rose BT. Anatomic study of retrobulbar needle path length. Ophthalmology. 1989;96:1221–4.
13. Riad W, Ahmed N. Single injection peribulbar anesthesia with a short needle combined with digital compression. Anesth Analg. 2008;107(5):1751–3.
14. Warner KJ, Cuschieri J, Jurkovich GJ, Bulger EM. Single-dose etomidate for rapid sequence intubation may impact outcome after severe injury. J Trauma. 2009;67(1):45–50.
15. Fields AM, Rosbolt MB, Cohn SM. Induction agents for intubation of the trauma patient. J Trauma. 2009;67(4):867–9.
16. Bruder EA, Ball IM, Ridi S, Pickett W, Hohl C. Single induction dose of etomidate versus other induction agents for endotracheal intubation in critically ill patients. Cochrane Database Syst Rev. 2015;1:7. https://doi.org/10.1002/14651858.CD010225.pub2.
17. Wadia S, Bhola R, Lorenz D, Padmanabhan P, Gross J, Stevenson M. Ketamine and intraocular pressure in children. Ann Emerg Med. 2014;64(4):385–8.
18. Drayna PC, Estrada C, Wang W, Saville BR, Arnold DH. Ketamine sedation is not associated with clinically meaningful elevation of intraocular pressure. Am J Emerg Med. 2012;30(7):1215–8.
19. Yoshikawa K, Murai Y. The effect of ketamine on intraocular pressure in children. Anesth Analg. 1971;50(2):199–202.
20. Mirakhur RK, Elliott P, Shepherd WF, Archer DB. Intra-ocular pressure changes during induction of anaesthesia and tracheal intubation. A comparison of thiopentone and propofol followed by vecuronium. Anaesthesia. 1988;43(Suppl):54–7.
21. Khosravi MB, Lahsaee M, Azemati S, Eghbal MH. Intraocular pressure changes after succinylcholine and endotracheal intubation: a comparison of thiopental and propofol on IOP. Indian J Ophthalmol. 2007;55(2):164.
22. Dillon JB, Sabawala P, Taylor DB, Gunter R. Action of succinylcholine muscles and intraocular pressure. Anesthesiology. 1957;18:44–9.
23. Lincoff HA, Breinin GM, Devoe AG. The effect of succinylcholine on the extraocular muscles. Am J Ophthalmol. 1957;43:440–4.
24. Vinik HR. Intraocular pressure changes during rapid sequence induction and intubation: a comparison of rocuronium, atracurium, and succinylcholine. J Clin Anesth. 1999;11(2):95–100.
25. Cook JH. The effect of suxamethonium on intraocular pressure. Anaesthesia. 1981;36(4):359–65.
26. Edmondson L. Intraocular pressure and suxamethonium. Br J Anaesth. 1997;79(1):146.
27. Wynands JE, Cromwell DE. Intraocular tension in association with succinylcholine and endotracheal intubation: a preliminary report. Can Anaesth Soc J. 1960;7:39–43.
28. Holloway KB. Control of the eye during general anaesthesia for intraocular surgery. Br J Anaesth. 1980;52(7):671–9.
29. Green K, Luxenberg MN. Consequences of eyelid squeezing on intraocular pressure. Am J Ophthalmol. 1979;88(6):1072–7.
30. Libonati MM, Leahy JJ, Ellison N. The use of succinylcholine in open eye surgery. Anesthesiology. 1985;62(5):637–40.
31. Vachon CA, Warner DO, Bacon DR. Succinylcholine and the open globe. Tracing the teaching. Anesthesiology. 2003;99(1):220–3.
32. Berkow L. Rapid Sequence Induction and Intubation (RSII) for anesthesia [internet]. Wolters Kluwer; c2017 [updated 2017 Aug 29]. Available from: https://www.uptodate.com/contents/rapid-sequence-induction-and-intubation-rsii-for-anesthesia.

33. Murphy DF. Anesthesia and intraocular pressure. Anesth Analg. 1985;64(5):520–30.
34. Bridion package insert. Merck Sharp & Dohme, a subsidiary of Merck & Co., Inc. Whitehouse Station, revised 06/2017.
35. Rocuronium package insert. Saint-Laurent: Hospira Healthcare Corporation; 2015.
36. Yi C, Jee D. Influence of the anasthetic depth on the inhibition of the oculocardiac reflex during sevoflurane anesthesia for pediatric strabismus surgery. Br J Anaesth. 2008;101(2):234–8.
37. Awad K, Ahmed H, Abushouk AI, Al Nahrawi S, Elsherbeny MY, Mustafa SM, Attia A. Dexamethasone combined with other antiemetics versus single antiemetics for prevention of postoperative nausea and vomiting after laparoscopic cholecystectomy: an updated systematic review and meta-analysis. Int J Surg. 2016;36(Pt A):152–63.
38. Kranke P, Eberhart LH, Roewer N, Tramèr MR. Pharmacological treatment of postoperative shivering: a quantitative systematic review of randomized controlled trials. Anesth Analg. 2002;94(2):453–60.
39. Mirakhur RK, Elliott P, Shepherd WF, McGalliard JN. Comparison of the effects of isoflurane and halothane on intraocular pressure. Acta Anaesthesiol Scand. 1990;34(4):282–5.

5 Diagnostic Imaging Considerations in Damage Control Ophthalmology

Aaron M. Betts and John L. Ritter

Ultrasound

Background

Ultrasound imaging uses piezoelectric crystals encased within a transducer probe to generate pulsed acoustic energy transmission through tissues. The piezoelectric crystals also respond to acoustic waves that return to the detector. Reflected and scattered echoes that return to the probe are used for image formation. Brightness mode (B-mode) imaging converts the amplitude of the returning echoes into a grayscale brightness that is displayed as a two-dimensional image on a display screen. Doppler imaging can also be used to evaluate flow, usually blood flow, by evaluating for Doppler frequency shifts [1].

Ultrasound imaging has several unique advantages that make it well-suited for ophthalmologic imaging. Sonography is a real-time, dynamic examination that provides detailed evaluation of more superficial structures. Unlike radiography and computed tomography (CT), ultrasound does not use ionizing radiation, making it safe and acceptable for use across all age ranges of patients. Modern ultrasound units are portable and can be brought to bedside or chairside at the point of care. The portability and versatility make ultrasound a highly available modality in austere humanitarian or combat environments. B-scan ultrasound is commonly performed in routine clinical practice. B-scan imaging is interpreted (and often performed) by the ophthalmologist. Therefore, the utility of this imaging modality is not limited to the availability of an interpreting radiologist. However, there are several limitations to ultrasound. The quality and diagnostic utility of ultrasound imaging are very operator dependent. Because bone is a very echogenic reflector, imaging through bony structures is problematic. However, this is usually not a significant limitation for the most common clinical applications of ophthalmic ultrasound. Ultrasonography is relatively contraindicated when there is suspicion of open globe injury, as the mechanical pressure of the applied probe can worsen injury.

Ocular ultrasound imaging is usually performed in routine clinical practice with dedicated ophthalmic "B-scan" units. These devices have a small probe and typically operate at frequencies ranging from 10 to 15 MHz. Ophthalmic imaging can also be performed with general diagnostic ultrasound units used in radiology departments and emergency rooms. Selection of a small footprint linear or curved array probe will allow visualization of ocular structures similar to a

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dedicated B-scan device. These transducers typically operate in frequency ranges of 5–15 MHz. Regardless of the device used, imaging is usually performed through a closed eyelid with ultrasound gel applied to the upper lid. In the intraoperative environment, ultrasound may be performed in direct contact with the anesthetized ocular surface [2].

Clinical Indications

The most common indication for ophthalmic ultrasound imaging is evaluation of posterior structures of the globe in a patient with opacified ocular media such as dense corneal opacity, severe corneal edema, hyphema, dense cataract, or vitreous hemorrhage. Ophthalmic ultrasound is useful in evaluation of vitreous hemorrhage, posterior vitreous detachment, and retinal detachment [3–9]. Ultrasound has also been described in detection of traumatic lens dislocation [3, 10, 11], evaluation of the integrity of the posterior capsule of the lens [12], and retrobulbar hemorrhage [13]. The use of ultrasound evaluation in ocular trauma, including military combat trauma has also been described in the medical literature [4, 14, 15]. However, this remains controversial due to the risk of worsening acute open globe injury [16]. Ultrasound can be used in detection of ocular foreign bodies, although sensitivity for detection is usually inferior to CT, and intraocular gas may mimic a foreign body [7, 17–19].

Plain Film X-Ray

Radiographic imaging, also called "X-ray" or "plain film," has been part of medical practice for more than a century. X-ray generation occurs in a vacuum sealed tube. A high voltage applied across the tube accelerates electrons toward an anode made of a high atomic number material (typically tungsten). The interactions of the accelerated electrons with the electron shells and the atomic nuclei of the anode result in the generation of high-energy photon. These photons (X-rays) are focused toward a detector while the patient is placed between the X-ray tube and the detector. The technology in X-ray generation and detection screens has greatly evolved since this imaging modality was first developed [1]. However, CT has largely replaced radiography as a diagnostic modality in the evaluation of ocular trauma. CT has a much greater sensitivity in the evaluation of the bony orbit and offers detailed evaluation of orbital soft tissues [20]. Nevertheless, in austere environments with limited or no access to CT, radiography can provide an evaluation of the bony structures of the face and orbit. Additionally, radiographs can evaluate for metallic or other radiopaque orbital foreign bodies [7, 21].

Computed Tomography (CT)

Background

The first clinically available CT scanner was released in 1972 and could only perform imaging of the head [1]. CT imaging technology has made significant advances in resolution, speed, and radiation dose. Clinical indications for CT have also greatly expanded and have become standard of care for civilian trauma. In the doctrine of the military medical evacuation system, CT first appears as an available imaging modality at role 3 facilities [22].

Computed tomography uses multiple rotationally acquired X-ray images and computer processing to generate multiplanar three-dimensional imaging. Similar to X-ray radiography, CT imaging is based upon the density of materials. Metal and bone will significantly attenuate an X-ray photon, whereas fat and gas have very little attenuation of X-ray photons. Fluid, muscle, connective tissue, and brain tissue have intermediate attenuation. The three-dimensional capability of CT does not improve the spatial resolution compared to X-ray radiography. However, the contrast resolution of CT is greatly expanded. The attenuation of materials on CT is described by the Hounsfeld unit (HU), with water designated as 0 HU. Attenuation values range from approximately −1000 to 3000. Fat and gas have negative

HU values, calcium 800 HU and higher, and metals greater than 1000 [1].

Clinical Indications

CT is well-suited for imaging in orbital trauma. CT imaging provides a detailed and rapid evaluation of osseous and soft tissue structures of the orbit. It is also sensitive in detection of retained ocular and orbital foreign bodies and their relationship to anatomic structures of the globe and orbit. In severe trauma, CT imaging will often be part of the initial trauma evaluation and is performed before ophthalmologic examination. Head CT imaging performed in conjunction with orbital imaging can also provide valuable information about intracranial extension of injury and coexistent brain injury (Fig. 5.1).

CT provides a detailed evaluation of the osseous structures in craniofacial trauma. CT is very sensitive in the detection of orbital wall fractures and can provide excellent definition of the extent of orbital wall injury, degree of fracture displacement, and relationship of fracture fragments to critical anatomic structures such as the extraocular muscles, optic nerve, and globe. CT is also extremely helpful in more complex facial/orbital fracture patterns such as naso-orbito-ethmoid (NOE), zygomaticomaxillary complex (ZMC), LeFort, and orbital apex fractures (Figs. 5.1 and 5.2).

Detection and localization of retained orbital foreign bodies can be critical in surgical planning and assessing risk of endophthalmitis or orbital cellulitis. While corneal and anterior chamber foreign bodies are easily detected by clinical examination, CT can detect foreign bodies in the deeper structures of the globe and orbit (Fig. 5.3). CT is also helpful in determining the depth of retained penetrating foreign objects and evaluating for penetration beyond the orbit. In military combat casualties, retained foreign bodies may be composed of metal, wood and other organic material, rocks and soil, glass, plastic, or bone fragments. The sensitivity for detection of foreign bodies depends upon size and composition. The sensitivity of detecting foreign bodies less than 0.06 mm^3 is approximately 65%, but approaches 100% above 0.06 mm^3 [7, 23, 24]. The sensitivity of detecting glass foreign bodies 0.5 mm in size has been reported at 48%, and increases to 96% at 1.5 mm [7, 24]. Plastic may also be hyperattenuating, although the imaging appearance with plastic is variable [7, 24, 25]. CT is also sensitive in the detection of small stones [26, 27]. CT has a lower sensitivity for detection of wooden foreign bodies (Fig. 5.4) as

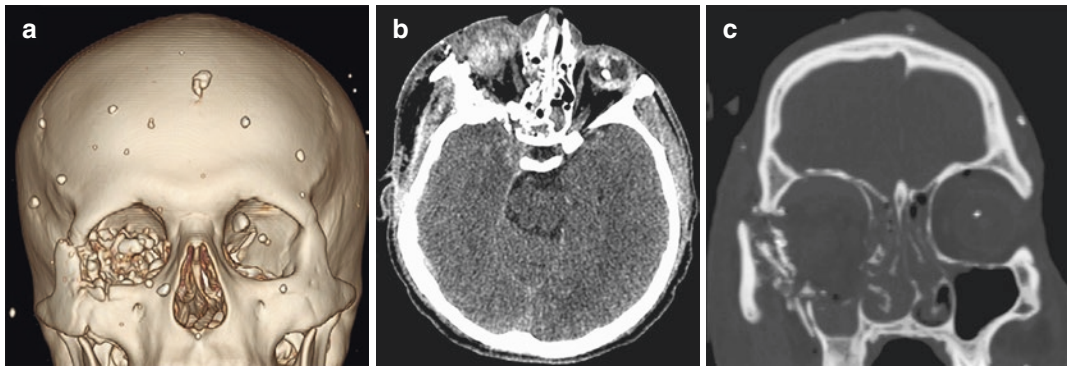

Fig. 5.1 Adult IED blast victim. (**a**) CT 3-D reconstruction. Multiple orbital and facial soft tissue foreign bodies. There are multiple facial fractures, to include right zygomaticomaxillary complex (ZMC) fracture pattern. (**b**) Axial CT soft tissue image through the level of the orbits and middle cranial fossa with bilateral open globe injuries with vitreous hemorrhage and metallic foreign body in the left vitreous. There are also right anterior temporal lobe parenchymal hemorrhages/contusions. (**c**) Coronal CT bone image showing complex facial fracture pattern and metallic foreign body in the left globe

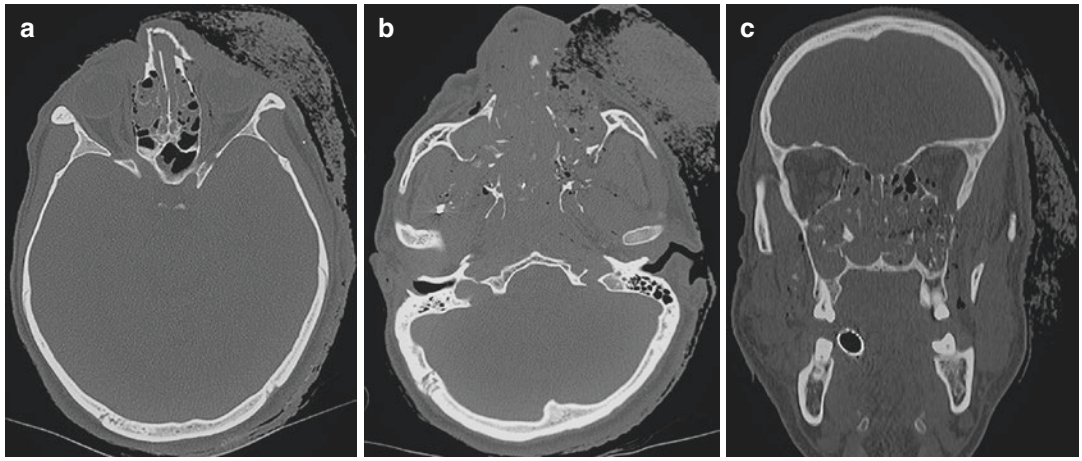

Fig. 5.2 Adult IED blast victim. CT bone images through the level of the orbits and maxillary sinuses. There are extensive facial and orbital fractures, to include medial wall and orbital floor fractures. (**a**) Axial CT bone image through the orbits. Comminuted nasal and medial orbital wall fractures. Facial and periorbital skin lacerations with bandage material in place. (**b**) Axial CT bone image through the maxillary sinuses. Comminuted maxillary sinus and nasal septal fractures. There is a metallic foreign body within the right masticator space. (**c**) Coronal CT bone image though the retrobulbar orbits and maxillary sinuses. Extensive facial and orbital fractures with orbital gas. Both globes were unruptured on ophthalmologic examination

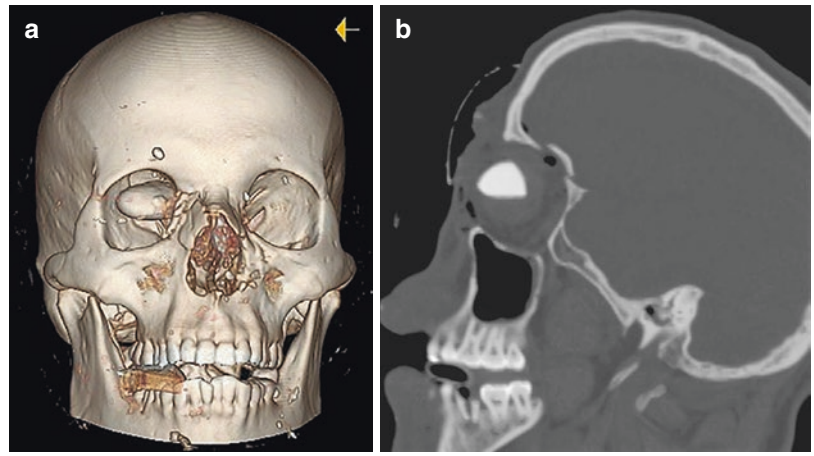

Fig. 5.3 Adult IED blast victim. Blast detonated near creek bed. (**a**) CT 3-D reconstruction with small superficial fragments and river rock embedded in right orbit. (**b**) Sagittal CT bone image with hyperattenuating stone within the right globe

the appearance of wood can mimic gas initially [7, 18, 28].

While clinical examination remains the gold standard for determining the integrity of the globe, there are CT findings that may indicate an open globe injury. These findings include foreign body or gas within the globe, dysmorphic appearance of the globe, (Fig. 5.5), or altered anterior chamber depth [24, 29–31]. The reported sensitivity for open globe injury by CT is variable, with sensitivity ranging from 56% to 76%, and specificity ranging from 85% to 100% [30, 32–35]. The sensitivity for anterior globe rupture has been reported up to 87% [36]. Alterations in anterior chamber depth (decreased in anterior rupture and increased in posterior rupture) have a sensitivity of 73% and specificity of 100% for asymmetry greater than 0.4 mm. Despite the high specificity and moderately high sensitivity of CT imaging for open globe injury, this should not replace clinical evaluation as a primary means of assessing globe integrity.

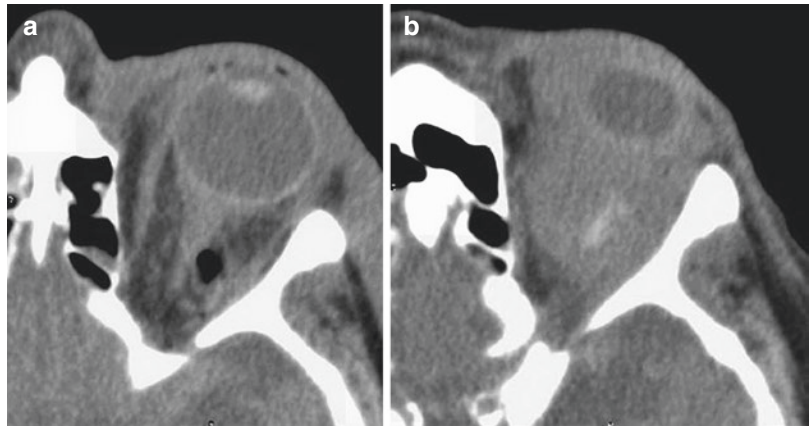

Fig. 5.4 Adult civilian non-combat injury. (**a**) Axial CT soft tissue image with low attenuating focus suspicious for orbital gas. Patient denies trauma, leaves emergency department and lost to follow-up. (**b**) Three months after initial injury patient returns with vision loss and pain. Axial CT soft tissue image with extensive orbital soft tissue attenuation/inflammatory change and central hyperattenuation in the region of prior low attenuation. Wood foreign body recovered at surgical exploration

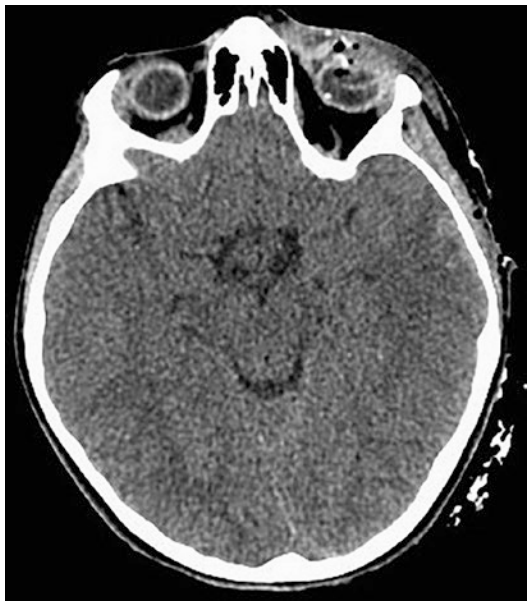

Fig. 5.5 Pediatric IED blast victim with open globe injury. Axial CT soft tissue image through the upper globes. There is a small metallic foreign body in the posterior left globe, anterior soft tissue gas, and deformation of the normal contour of the globe. Globe was perforated on ophthalmologic examination. There was also intracranial hemorrhage (partially visualized in left temporal lobe), perforating fragments of the torso and extremities, and extensive bone and soft tissue injury of the hand (not shown)

Practical Considerations

In order to maximize the effectiveness of CT imaging in a combat environment, several elements of care are necessary. A trained CT technologist is critical for optimal patient positioning, radiation dose management, image optimization, and image processing. A radiologist with training and expertise in trauma imaging is also extremely valuable in evaluating the clinically apparent ophthalmic injuries beyond the face and orbits, as well as clinically occult injuries that can be overshadowed by the more obvious distracting injuries. Multiplanar and three-dimensional reconstruction capabilities (Figs. 5.1a and 5.3a) can greatly enhance the quality and speed of radiologist interpretation and enhance the radiologist's ability to convey significant findings to the trauma team and operative surgical team.

Magnetic Resonance Imaging (MRI)

Background

Magnetic resonance (MR) imaging uses a strong magnetic field and radiofrequency (RF) waves to interrogate the properties of water protons in

biological tissues. In the strong magnetic field, the magnetic moments of hydrogen protons rotate or precess (like a spinning top) at a specific angular frequency, with the average magnetic moment aligned to the main magnetic field. Upon exposure to an RF signal at resonant frequency, the magnetic moments of the precessing protons deflect toward the transverse plane. After the RF signal is turned off, the magnetic moments of the deflected protons will recover back towards their original state of alignment with the main magnetic field. The rate of recovery is an exponential decay, described by the T1 time constant. The deflected magnetic moments precess in phase when they are first deflected by the RF signal. After the RF signal is turned off, the deflected magnetic spins will begin to dephase. The rate of dephasing is also an exponential decay, described by the T2 time constant. The density of available protons varies in biological tissues, and the T1 and T2 properties are variable in different tissue types. Complex sequences of RF pulses and magnetic gradients interrogate these properties of tissues and spatially encode the information, allowing for image formation with excellent contrast resolution between tissue types [1, 37].

Clinical Indications

The role of MR imaging in acute injury is extremely limited. In combat-related trauma, in which there is suspicion for retained metallic foreign bodies, MR is usually contraindicated. Retained metallic foreign bodies can undergo translational or rotational movement in the high magnetic field environment due to either static magnetic field or due to magnetic field gradients. Metallic objects may also induce heating due to RF current induction. In the eye and orbit, movement and heating of retained metallic foreign bodies can result in significant injury and vision loss. The one clinical scenario in which MR may be more sensitive than CT is in detection of wooden foreign bodies. The CT attenuation of wood mimics gas. However, the measured CT attenuation of wood ranges from approximately −200 to −100 HU (Fig. 5.5), while gas measures approximately −1000 HU. As an organic material, wood foreign bodies elicit a strong inflammatory reaction, and are associated with a high risk of endophthalmitis and orbital cellulitis. MR may be more sensitive at detecting the early inflammatory changes associated with wood foreign bodies [7, 18, 28].

The availability of MR imaging in combat environments and humanitarian missions is usually extremely limited. The U.S. Department of Defense deployed several MR scanners to Afghanistan in 2011 for both clinical use as well as data collection for traumatic brain injury research [38, 39]. The scanners were later decommissioned and removed from theater in 2014 [40]. The availability of MR imaging in humanitarian assistance outside the USA will depend largely upon the regional availability of an MR scanner in the host nation.

Summary

The availability and portability of diagnostic imaging as an adjunct in damage control ophthalmology is variable across imaging modalities and different environments of care. Ultrasound imaging is among the most portable imaging modalities and can be performed at the point of care or intraoperatively. Plain film X-ray imaging is widely available, but usually requires a hospital or clinic setting, and may have limited utility in damage control ophthalmologic care. Computed tomography (CT) is widely available across most US hospitals. However, CT is more costly, and the availability in austere environments is more limited. Magnetic resonance imaging (MRI) is even more costly. Due to technical, safety, and maintenance requirements of MR scanners, the availability of MRI in austere environments is usually not feasible.

References

1. Bushberg JT, Seibert JA, Leidholdt EM, Boone JM. The essential physics of medical imaging. 2nd ed. Philadelphia: Lippincott Williams & Wilkins; 2002.

2. Shiraki N, Wakabayashi T, Sato T, Sakaguchi H, Nishida K. Intraoperative B-scan ultrasonography and pars plana vitrectomy for severe open globe injury with hemorrhagic retinal and choroidal detachment. Graefes Arch Clin Exp Ophthalmol. 2017;255(11):2287–91.
3. Frasure SE, Saul T, Lewiss RE. Bedside ultrasound diagnosis of vitreous hemorrhage and traumatic lens dislocation. Am J Emerg Med. 2013;31(6):1002 e1–2.
4. Kwong JS, Munk PL, Lin DT, Vellet AD, Levin M, Buckley AR. Real-time sonography in ocular trauma. AJR Am J Roentgenol. 1992;158(1):179–82.
5. McNicholas MM, Brophy DP, Power WJ, Griffin JF. Ocular trauma: evaluation with US. Radiology. 1995;195(2):423–7.
6. Scott IU, Smiddy WE, Feuer WJ, Ehlies FJ. The impact of echography on evaluation and management of posterior segment disorders. Am J Ophthalmol. 2004;137(1):24–9.
7. Sung EK, Nadgir RN, Fujita A, Siegel C, Ghafouri RH, Traband A, et al. Injuries of the globe: what can the radiologist offer? Radiographics. 2014;34(3):764–76.
8. Vrablik ME, Snead GR, Minnigan HJ, Kirschner JM, Emmett TW, Seupaul RA. The diagnostic accuracy of bedside ocular ultrasonography for the diagnosis of retinal detachment: a systematic review and meta-analysis. Ann Emerg Med. 2015;65(2):199–203. e1
9. Yoonessi R, Hussain A, Jang TB. Bedside ocular ultrasound for the detection of retinal detachment in the emergency department. Acad Emerg Med. 2010;17(9):913–7.
10. Boniface KS, Aalam A, Salimian M, Liu YT, Shokoohi H. Trauma-induced bilateral ectopia lentis diagnosed with point-of-care ultrasound. J Emerg Med. 2015;48(6):e135–7.
11. Eken C, Yuruktumen A, Yildiz G. Ultrasound diagnosis of traumatic lens dislocation. J Emerg Med. 2013;44(1):e109–10.
12. Tabatabaei A, Hasanlou N, Kheirkhah A, Mansouri M, Faghihi H, Jafari H, et al. Accuracy of 3 imaging modalities for evaluation of the posterior lens capsule in traumatic cataract. J Cataract Refract Surg. 2014;40(7):1092–6.
13. Kniess CK, Fong TC, Reilly AJ, Laoteppitaks C. Early detection of traumatic retrobulbar hemorrhage using bedside ocular ultrasound. J Emerg Med. 2015;49(1):58–60.
14. Moreno L, Velasquez LF, Restrepo CA, Paulo JD, Donado J, Munoz ML, et al. Ocular trauma from land mines among soldiers treated at a University Hospital in Medellin, Colombia. Colomb Med (Cali). 2013;44(4):218–23.
15. Ritchie JV, Horne ST, Perry J, Gay D. Ultrasound triage of ocular blast injury in the military emergency department. Mil Med. 2012;177(2):174–8.
16. Debiec M, Frazier T, Colyer M, Nelson M. Inappropriate use of ultrasound in ocular trauma. Mil Med. 2012;177(12):v–vi; author reply vi.
17. Fielding JA. The assessment of ocular injury by ultrasound. Clin Radiol. 2004;59(4):301–12.
18. Modjtahedi BS, Rong A, Bobinski M, McGahan J, Morse LS. Imaging characteristics of intraocular foreign bodies: a comparative study of plain film X-ray, computed tomography, ultrasound, and magnetic resonance imaging. Retina. 2015;35(1):95–104.
19. Shiver SA, Lyon M, Blaivas M. Detection of metallic ocular foreign bodies with handheld sonography in a porcine model. J Ultrasound Med. 2005;24(10):1341–6.
20. Kennedy TA, Corey AS, Policeni B, Agarwal V, Burns J, Harvey HB, et al. American College of Radiology Appropriateness Criteria: oribts, vision and vision loss. 2017. Available from: https://acsearch.acr.org/docs/69486/Narrative/.
21. Saeed A, Cassidy L, Malone DE, Beatty S. Plain X-ray and computed tomography of the orbit in cases and suspected cases of intraocular foreign body. Eye (Lond). 2008;22(11):1373–7.
22. Emergency War Surgery. Fourth United States Revision ed. Fort Sam Houston: Borden Institute; 2013.
23. Mester V, Kuhn F. Intraocular foreign bodies. Ophthalmol Clin N Am. 2002;15(2):235–42.
24. Pinto A, Brunese L, Daniele S, Faggian A, Guarnieri G, Muto M, et al. Role of computed tomography in the assessment of intraorbital foreign bodies. Semin Ultrasound CT MR. 2012;33(5):392–5.
25. Duker JS, Fischer DH. Occult plastic intraocular foreign body. Ophthalmic Surg. 1989;20(3):169–70.
26. Arnaiz J, Marco de Lucas E, Piedra T, Torres M, Blanco G, Gonzalez-Mandly A, et al. Intralenticular intraocular foreign body after stone impact: CT and US findings. Emerg Radiol. 2006;12(5):237–9.
27. Lakits A, Prokesch R, Scholda C, Bankier A. Orbital helical computed tomography in the diagnosis and management of eye trauma. Ophthalmology. 1999;106(12):2330–5.
28. Tas S, Top H. Intraorbital wooden foreign body: clinical analysis of 32 cases, a 10-year experience. Ulus Travma Acil Cerrahi Derg. 2014;20(1):51–5.
29. Dunkin JM, Crum AV, Swanger RS, Bokhari SA. Globe trauma. Semin Ultrasound CT MR. 2011;32(1):51–6.
30. Joseph DP, Pieramici DJ, Beauchamp NJ Jr. Computed tomography in the diagnosis and prognosis of open-globe injuries. Ophthalmology. 2000;107(10):1899–906.
31. Sevel D, Krausz H, Ponder T, Centeno R. Value of computed tomography for the diagnosis of a ruptured eye. J Comput Assist Tomogr. 1983;7(5):870–5.
32. Arey ML, Mootha VV, Whittemore AR, Chason DP, Blomquist PH. Computed tomography in the diagnosis of occult open-globe injuries. Ophthalmology. 2007;114(8):1448–52.
33. Chou C, Lou YT, Hanna E, Huang SH, Lee SS, Lai HT, et al. Diagnostic performance of isolated orbital CT scan for assessment of globe rupture in acute blunt facial trauma. Injury. 2016;47(5):1035–41.
34. Hoffstetter P, Schreyer AG, Schreyer CI, Jung EM, Heiss P, Zorger N, et al. Multidetector CT (MD-CT)

in the diagnosis of uncertain open globe injuries. Rofo. 2010;182(2):151–4.
35. Yuan WH, Hsu HC, Cheng HC, Guo WY, Teng MM, Chen SJ, et al. CT of globe rupture: analysis and frequency of findings. AJR Am J Roentgenol. 2014;202(5):1100–7.
36. Gad K, Singman EL, Nadgir RN, Yousem DM, Pillai JJ. CT in the evaluation of acute injuries of the anterior eye segment. AJR Am J Roentgenol. 2017;209(6):1353–9.
37. Westbrook CW, Roth CJ, Talbot J. MRI in practice. 4th ed. West Sussex, : Wiley-Blackwell; 2011.
38. First mobile MRI systems to be sent to Afghanistan theater. 2011. Available from: http://www.navy.mil/submit/display.asp?story_id=60894.
39. Afghanistan theater receives state-of-the-art MRI systems. 2011. Available from: http://www.navy.mil/submit/display.asp?story_id=63216.
40. Zoroya G. MRI machines for treating soldiers pulled from war zone. 2014. Available from: https://www.usatoday.com/story/news/nation/2014/01/18/tbi-brain-mri-mullen-blast/4489913/.

Damage Control Surgery: Blast—Anterior Segment Trauma

6

Anthony J. Johnson, J. Richard Townley III, and Joseph F. Pasternak

Prehospital

The improvised explosive device (IED), the signature weapon of the Iraqi and Afghanistan theaters of conflict, contributed over 93% of the ocular injuries encountered by deployed ophthalmologists at the height of the hostilities [1]. Additionally, in the wake of the Boston Marathon, and Oklahoma City bombings, its use has found its way into urban society. Unlike typical civilian injuries, in which victims tend to be injured individually, the emergence of the IED now meant that patients often presented in groups or in mass casualty situations. Explosions from IEDs result in a rapid rise in barometric pressure in the vicinity of the explosions leading to ocular damage from the blast overpressure [2]. More significant are the secondary blast effects caused by foreign bodies picked up by the blast and propelled by the blast overpressure. For example, the Baghdad United Nations office building in 2003 was attacked by an automotive IED. This attack resulted in eight patients with ruptured globes, three of which were bilateral. They all presented simultaneously to the Combat Support Hospital outside of Baghdad for surgical evaluation and treatment.

Due to the advances in patient evacuation and communication, it is not uncommon for the emergency room at the receiving facility, far from the initial point of injury, to receive a report of the injured soldier well in advance of their arrival. Good communication between the receiving ophthalmologist and the emergency providers may facilitate improved patient outcomes, by reiterating the need to protect the eye with a Fox shield and ensuring the patient receives broad-spectrum antibiotics at the point of injury, en route, or immediately upon arrival at your facility. The practice of early administration of antibiotics, combined with the sterilizing effect of the blast, is felt to largely explain the near absence of endophthalmitis in IED blast victims with intraocular foreign bodies (IOFBs). Only four cases of endophthalmitis are reported in over 700 casualties with intraocular foreign bodies, and 0 cases in the 79 eyes with IOFB's removed at WRAMC between 2003 and 2005 [3].

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Initial Evaluation

Depending on the mechanism of injury, clinical signs that are predictive of ocular rupture and the need for evacuation include vision of light perception or worse, and afferent pupillary defect, hemorrhagic chemosis, a very shallow or very deep anterior chamber, or a tented pupil [4–6]. If these signs are not clearly noted, due to the varied composition of the materials in the devices (sand, wood, and glass), the evaluation of IED victims can present a diagnostic challenge. In addition to the standard eight-part eye examination, imaging studies play an important role in pre-surgical planning. Personal observation supported by numerous studies indicates that one should strongly consider the use of ultrasound (being cautious not to exert any pressure on a suspected ruptured globe), in addition to direct observation, and the computerized tomography (CT) in the initial evaluation of blast victims, especially when homemade devices are used. Wood fragments are often not seen on the CT scan or plain radiographs. This is especially true if the wood is situated near muscles or bone [7]. Additionally, if the victim was in the direct vicinity of the explosion, thermal injuries would be seen in addition to the traditional blunt and penetrating injuries caused by the explosive projectiles (Fig. 6.1a, b).

When mass casualty situations occur, there is often a desire to quickly get patients triaged, evaluated, and to the operating room. As one plans the initial evaluation of patients, a few considerations that are common in times of conflict and need to be considered as a surgical plan are developed. First, bilateral injuries are common, and when the mechanism of injury is blast, non-contiguous. During the Iraqi conflict 22–37% of ocular injuries were bilateral, and 80% involved a combined globe, oculoplastic, and neuro-ophthalmic injuries [8]. Meticulous evaluation of both globes for subtle injuries is a must. Second, ocular injuries do not occur in isolation. Eight-five percent of patients evacuated with ocular injuries will have associated conventional injuries [9]. These injuries can also be life-threatening. With careful consultation with the head of triage, the priority of all injuries needs to be determined, especially when the patient is being evacuated from another hospital in-theater with non-subspecialty surgical capability. It may be necessary for other significant injuries, such as large unstable fractures, or vascular injuries to be stabilized at the first facility prior to evacuation to a surgical subspecialty hospital. Failure to coordinate care can unnecessarily delay surgical repair of the eyes once the patient reaches your facility. In rare circumstances, simultaneous surgery can be performed. The feasibility of this approach may be dictated by your operating room size and facilities, and the ability of the other specialty surgeon to minimize movement of the patient for your microsurgery. During the initial

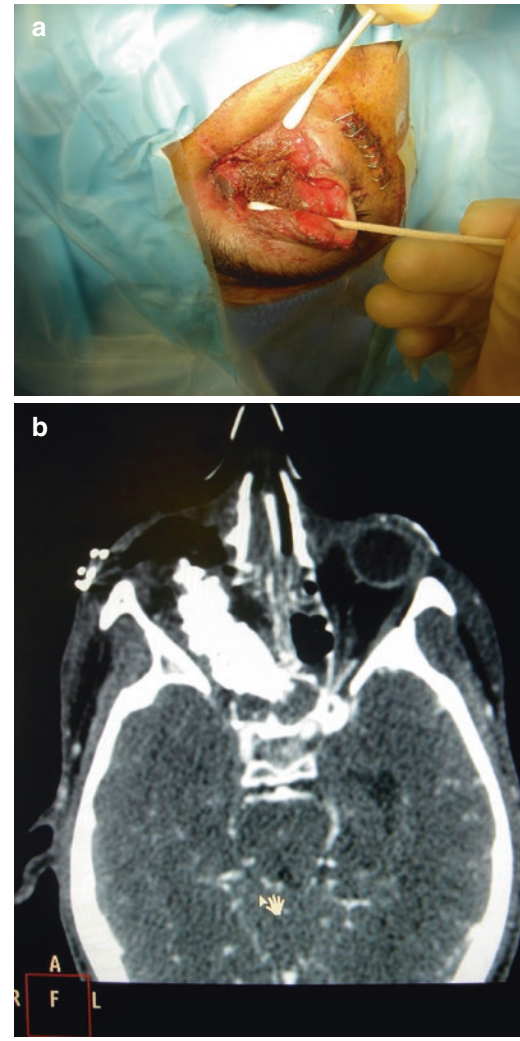

Fig. 6.1 (**a**) Photo of mud injected into the orbit by an IED. (**b**) CT scan of the same patient. (Courtesy of David Zumbro, MD)

3 months of Operation Iraqi Freedom, a statistical evaluation of the injured found that 35% of the intracranial foreign bodies removed by the neurosurgeons entered the cranial vault through the orbit [10], again underscoring the importance of polytrauma evaluations, including imaging, when approaching blast victims.

Finally, even at outside temperatures greater than 100°, a patient with significant blood loss can become hypothermic. The body loses heat from hemorrhage, and from evaporation of sweat. Convection of air as it passes over the body during transport likewise can cool the body surface. Poor muscle perfusion from hemorrhage, coupled with low blood pressure, reduces the influx of oxygen and glucose to the muscle and fat mitochondria. Thus the body's chief heat-generating mechanisms are inhibited while heat loss continues, resulting in a drop in body temperature [11]. This is very important because patients who are hypothermic do not coagulate normally [12]. They develop temperature induced reversible inhibition of their platelet activation which improves when their temperature returns to 37°C [12]. During peacetime these details are typically addressed by the trauma service. In mass casualty situations, the desire to initiate treatments can shorten the evaluation process. Failing to recognize and correct low body temperature prior to proceeding to the operating room can result in significant patient bleeding and complicate the surgical correction of eye injuries.

Surgical Interventions

Superficial Foreign Bodies

Due to the nature in which improvised explosive devices are employed, and the tremendous achievements in prehospital care, it is not uncommon for victims in the immediate vicinity of an explosion to survive. These patients frequently present with gun powder and mud injected into the facial tissues, and hundreds of small foreign bodies injected into the cornea and eyes without a concomitant rupture of the globe. In these cases, in addition to a complete examination to rule out an underlying rupture, these patients may need repeated trips to the operating room to meticulously irrigate and express the mud, sand, glass, and powder out of the face and ocular surface. It is strongly recommended this be accomplished as close to the time of the original injury as possible. First, the openings in Bowman's membrane are fresh, facilitating access for deep foreign bodies which can minimize the damage inherent in late removal of the foreign bodies. Delaying the initial debridement may result in the overlying defect sealing, secondary scar formation larger than the original injury, unnecessary reduction in vision, additional discomfort to the patient as sealed wounds need to be reopened, or multiple trips to the ophthalmologist as superficial foreign bodies migrate to the surface of the cornea. Not all foreign bodies need to be removed; however, a concerted effort should be made to remove the significant foreign bodies that operational situation allows. Failure to remove the dirt and gun powder can result in disfiguring scarring and tattooing of the overlying skin and cornea [13].

In cases in which there is a concomitant ruptured globe, care must be taken to protect the eye with a Fox shield before, and during your preoperative evaluation, with the foreign body removals performed at the time of globe repair.

Anesthesia

Due to the risk of exacerbating ocular injuries with large volumes of intraorbital anesthetic putting pressure on the globe, retrobulbar blocks are contraindicated in patients with open globes. It is important that the patient be placed under general anesthesia. Placing a wounded and unconscious patient under general anesthesia is not without significant risks. Often the anesthesia provider is unable to obtain any history from the patient. In such circumstances, a rapid sequence induction is frequently performed to minimize the chance of aspiration. Within the anesthesia literature, there is controversy with regard to the use of succinylcholine for the rapid sequence induction [14]. It is known to increase intraocular pressure (IOP) and also that contracture of the extraocular muscles during the induction can theoretically exacerbate intraocular injuries. Equally important is

the fact that many anesthesia providers deployed to combat may not yet have encountered a patient with a ruptured globe in their clinical practice. Attempting to avoid the use of succinylcholine in order to prevent involuntary contracture of the extraocular muscles, and switching to an induction regimen with which the provider is not comfortable, can result in the patient not obtaining an initial quick and deep induction. Irritability of the airway during this circumstance can result in coughing on the tube, raised intraocular pressure, and definite exacerbation of preexisting injuries. Thus it is recommended that the ophthalmologist communicate with the anesthesia provider prior to surgery and communicate the need for a smooth deep intubation and extubation by the provider's method of choice, rather than attempting to avoid succinylcholine use [14].

Once the patient is under anesthesia, all wounds of the face need to be meticulously irrigated, and all foreign material debrided from the wounds. Gunpowder needs to be removed from the skin. The ocular surface is meticulously evaluated to determine the size and architecture of any wounds that are noted. If a cornea laceration is noted and the surgeon is unsure whether a wound is full thickness, a Siedel test can be performed. Partial-thickness wounds that do not have irregular contour can be treated with a bandage contact lens or pressure patch. If the wound is full thickness and self-sealing, it likewise may be considered for conservative treatment; if it is <2 mm in length and the patient can refrain from Valsalva or straining while the wound heals, likewise conservative management is reasonable. Lacerations 3mm or greater should be closed with a suture. If the nature of the patients job or lifestyle prohibits the patient from avoiding heavy lifting or bending (ie works as a first responder), then their wounds should be closed with a suture. All knots should be trimmed and buried superficially in the cornea away from the visual axis [15].

General Suturing Principles

Once the suturing process is initiated a number of important principles have to be followed to ensure a watertight, low astigmatic wound. Due to the inherent stiffness of the cornea great accuracy in placement of the sutures is critical. To take advantage of the lamellae design of the cornea collagen, all sutures should be placed perpendicular to the wound. Obliquely placed sutures will result in vector forces that will shift the wound laterally [16]. Perpendicular placement will ensure the suture is able to exert the most effective compression across the wound.

On wounds that are completely vertical through the tissue, to ensure optimum tissue alignment, one must ensure that the suture enters and exits the tissue equal distance from the laceration, and that the suture crosses the laceration within the tissue at equal depths and as close to Descemet's membrane as possible (Fig. 6.2a) [17]. If the suture pass on one side is too long, the tissue will override (Fig. 6.2b, c).

As the simple interrupted suture tightens it will redistribute forces equally in every direction. Too shallow a suture pass will result in the lower portion of the wound gaping, increasing the risk of tissue override and leakage (Fig 6.2c) [18].

Unequal depths will result in tissue override and again increased risk of leakage (Fig 6.2c). Sutures that are placed too tight will result in the wound either everting or inverting, increasing astigmatic risk (Fig. 6.3).

As a suture is tightened within the cornea, it compresses the tissues. The magnitude of the compression dissipates away from the suture in a linear fashion reaching zero at a distance equal to the length of the suture. This area is a called the zone of compression. Thus longer sutures have larger and longer zones of compression and conversely shorter suture passes have smaller zones of compression. To ensure a watertight closure, one must ensure the zones of compression from adjacent sutures overlap [19]. This will ensure a compression force exists across the length of the wound sufficient to overcome physiologic intraocular pressure. The higher the intraocular pressure, the more stress across the wound, the more extensive the overlap in compression zones is required (Fig. 6.4). Under most circumstances with an open globe, extensive overlap is not an issue; however, when an open globe is combined with extensive posterior chamber hemorrhage, extensive compression zone overlap may be required [19].

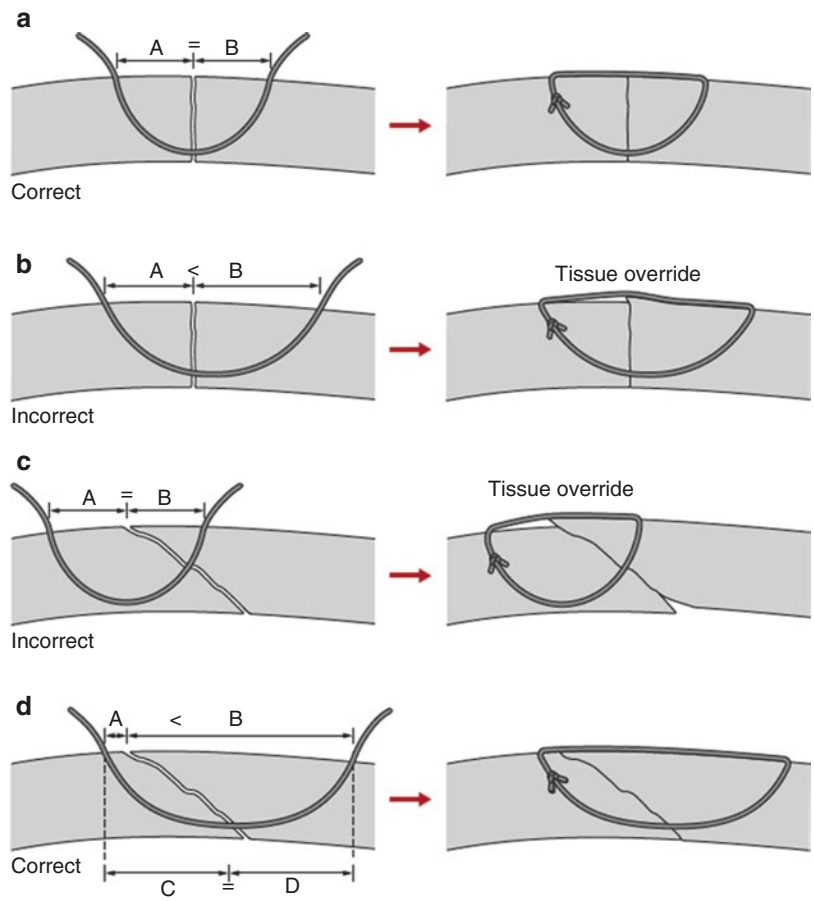

Fig. 6.2 (**a**): Vertical Laceration: Sutures currently placed with suture the same depth on both sides of the wound, and entry and exit points equally spaced with regards to the deep portion of the wound. (**b**): Incorrect suture placement, segment B, exceeds segment A, resulting in tissue override. (**c**): Incorrect suture placement, the sutures are equally spaced with reference to the surface of the wound, not the deep portion, resulting in tissue override. (**d**): Oblique Laceration: Sutures currently placed with suture the same depth on both sides of the wound, and entry and exit points equally spaced with regards to the deep portion of the wound

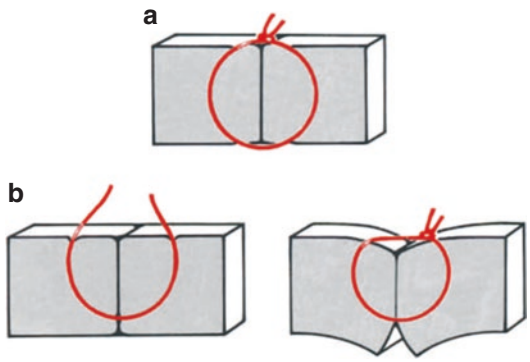

Fig. 6.3 Superficially placed overtightened sutures in the cornea cause non enclosed portions of the wound to gape. This wound gape can facilitate wound leakage as intraocular pressure rises

In the event of tissue loss, or shredded tissue in which a watertight seal cannot be obtained, adjuvants such as a transparent gamma-irradiated cornea (visiongraft), which have a shelf life of up to 2 years [20], can be ordered to size, cut to various sizes using either small trephines or free handed techniques, is an ideal material to repair non-sutureable corneal and scleral wounds, and can be stored in your operating room or brought in bulk for your deployment. Additionally, banked sclera, cyanoacrylate glue, tutoplast, conjunctival pedicle flaps, or other wound filling material can be used (Figs. 6.5a, b and 6.6) [21].

Incarcerated Iris

When the globe is lacerated within 3 mm of the limbus, the change in pressure is often accompanied by the iris incarcerating itself in the wound, thereby creating a watertight seal. As long as the pressure gradient across the wound persists, the flexible iris will be pushed out of the wound, recreating the watertight seal. The

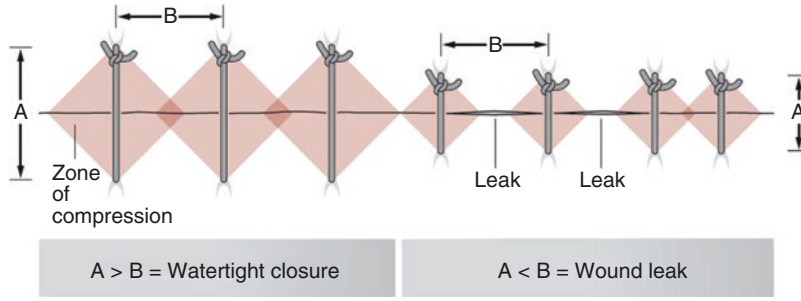

Fig. 6.4 Zones of compression. Different lengths of suture bites result in different zones of compression. When the zones of compression overlap, adequate wound closure is achieved (arrows). (Reused with permission © Springer Nature 2007 [19])

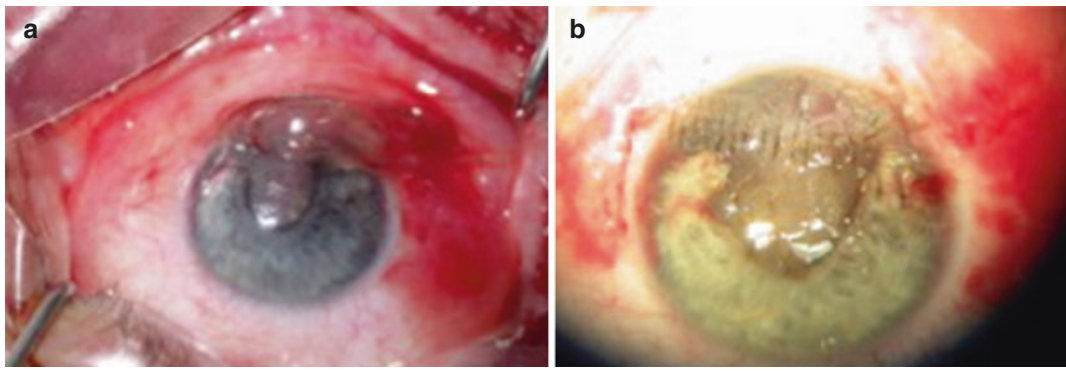

Fig. 6.5 (**a**) Ruptured globe with posterior segment hemorrhage resulting in the retina being expulsed out of the eye. (**b**) Surgical closure required significant overlapping of the compression zones due to the high intraocular pressure

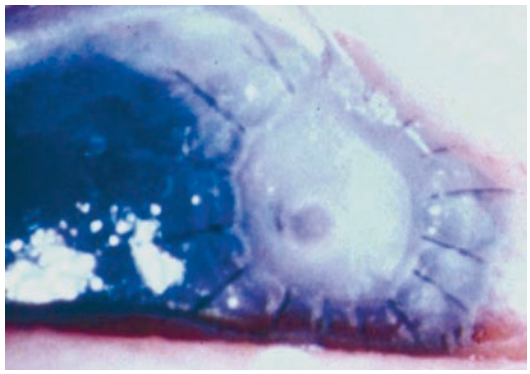

Fig. 6.6 The area around the wound is marked with a trephine, and a partial-thickness bed is created with lamellar dissection. An autologous same-size lamellar piece of tissues is harvested from a separate area of the same eye and secured into position with interrupted sutures. (Reused with permission © Springer Nature 2007 [21])

prolapsed iris should be carefully examined. If the iris has been incarcerated for greater than 24 hours, strong consideration should be made to excising it to reduce the risk of endophthalmitis. Additionally, if the iris appears non-viable, the portion of the iris that is outside of the wound should be excised [22].

Simply repositing the iris will result in repeated egress of the iris out of the wound due to the pressure gradient across the wound. To overcome this situation, a few principles need to be employed. First, obtain control of the anterior chamber by placing a paracentesis port. The paracentesis should be placed in a fashion that facilitates sweeping the iris from the wound without requiring the instrument to pass directly across the visual axis. Failure to do so could

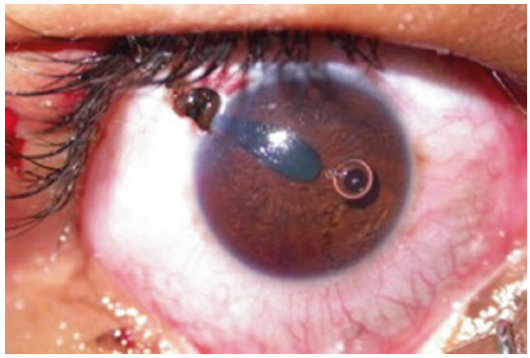

Fig. 6.7 Anterior segment laceration by a multi-function tool. The incarcerated iris results in a peaked pupil pointing toward the injury

result in lenticular damage if the chamber collapses suddenly while sweeping iris out of the wound. Second, place a miotic such as Miochol or Miostat into the anterior chamber if it is available. The miotic will contract the iris and facilitate reposition of the uvea. Place a small amount of viscoelastic into the anterior chamber to protect the endothelium, then slowly remove fluid from the anterior chamber to decrease the gradient across the wound. This will allow the surgeon to reposit the iris without the iris immediately coming out of the wound. Once the iris can safely be reposited into the anterior chamber, place the first sutures to re-approximate the wound (under low intraocular pressure). Slowly add additional sutures. One can then slowly inflate the interior chamber and place the rest of the sutures safely without damaging the iris (Fig. 6.7).

Stellate Lacerations

Stellate lacerations of the cornea are often very difficult to close. Depending on the complexity of the laceration, multiple surgical techniques may be successfully employed. The most important initial consideration is to ensure proper alignment of the tissue. Next, the peripheral aspects of the legs of the laceration are closed. As the laceration is closed, one can observe how effectively the central portion of the laceration re-approximates. If the edges of the laceration are not extensively shelved, then additional compression of the center can be obtained via simple interrupted or mattress sutures across the central portions of the laceration. Two additional methods that are variations of a purse-string suture may be used: Eisner method (Fig. 6.8a) and the Akkin method (Fig. 6.8b). The Eisner method is best facilitated with the use of a diamond blade which can make the small incisions between the legs of the stellate laceration when the intraocular pressure is very low [23].

In the event of a cornea-sclera laceration, the limbus is closed first with 9-0 nylon suture, then the cornea is repaired with 10-0 nylon, followed by the sclera with 8-0 nylon suture.

The use of vicryl suture is discouraged, because scleral wounds were found to reopen during secondary retina repair after patients were evacuated to Walter Reed Army Medical Center during Operation Iraqi Freedom, significantly complicating secondary reconstructive surgeries.

Any laceration that crosses the limbus should be investigated to its fullest extent. If the laceration passes through or underneath a rectus muscle, the muscle will need to be dis-inserted; the laceration will then be explored and repaired. Then the rectus muscle should be replaced.

Lacerations should be closed to the fullest extent possible. Very posterior lacerations will lead to fibrosis and close over a 7- to 10-day period.

Scleral Lacerations

Scleral lacerations represent a unique challenge in closure, as the extent of the laceration is not always visible. Additionally with blunt trauma, the sclera has a propensity to rupture in a long shelved fashion. Careful inspection of the scleral thickness and architecture is necessary to ensure proper alignment of the sclera in these situations.

Any exposed vitreous present should be removed prior to repositing of uveal tissue. This can be accomplished with a Wexcell sponge. The vitreous will strongly adhere to the

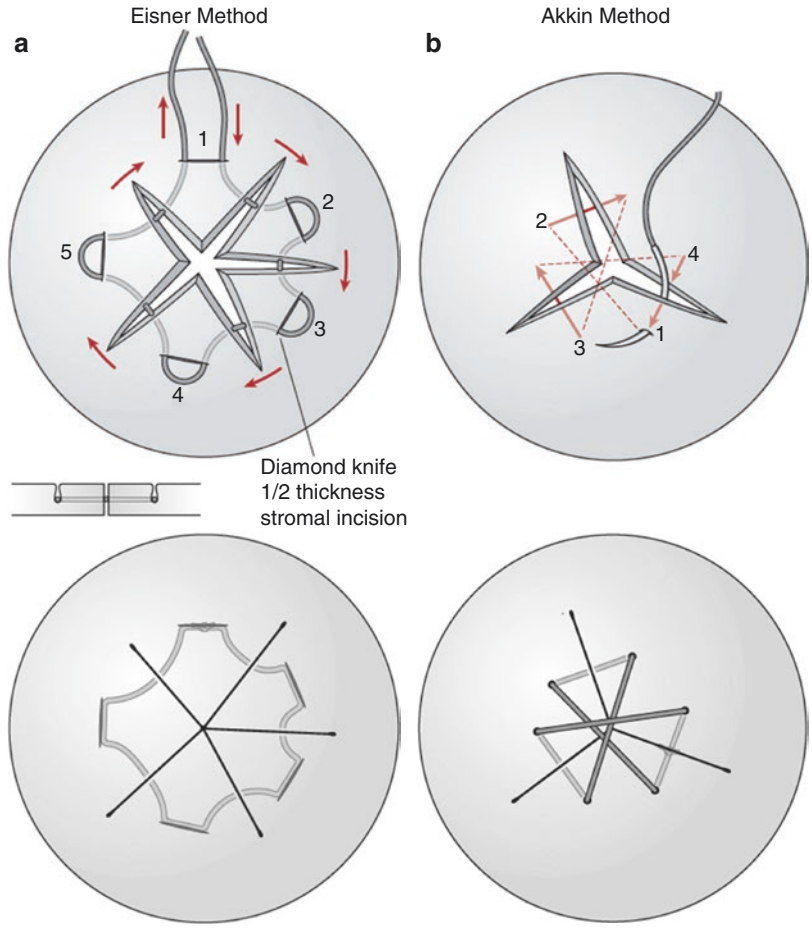

Fig. 6.8 (**a**) In the Eisner method, a partial-thickness incision is made between the arms of the laceration and a purse-string suture is passed through these grooves and tightened to approximate the apices of the wound. Overtightening of the purse-string suture will result in forward displacement of the apices and wound leakage. The suture is buried when it is tied and left in place indefinitely. (**b**) In the Akkin method, no partial-thickness groove is made. The suture is passed through the tissue and over the apices of the wound to appose the tissue. (Reused with permission © Springer Nature 2007 [23, 24])

wexcell sponge. The surgeon can then touch the vitreous with the wexcell sponge to gently grasp the vitreous, followed by trimming the vitreous with a Vanness scissor, or the use of a vitrector. After the vitreous is removed the uveal contents can be addressed. If the surgeon has a skilled assistant, the surgeon can place the suture through the distal wound, the assistant can then gently reposit the tissue with a spatula, and then the surgeon can place a suture through the proximal wound edge slowly tightening the suture over the spatula as it is gently removed. If the surgeon is operating alone or does not have a skilled assistant, then I have found a self-burying knot technique to be the most effective. The surgeon everts the proximal edge of the wound and uses the posterior surface of the needle to gently push the uvea away from the wound edge as the first half of the suture is passed. The distal wound edge is then everted and the needle is passed through the distal portion of the wound with care not to incorporate the uveal tissue to the point that the tip of the needle is just exiting the sclera. If the suture is passed perpendicular to the scleral surface, it will remain vertical in the tissue when released. The surgeon can then gently reposit the tissue and advance the needle over the uvea. The initial throws of the suture are then tied loosely. The skilled surgeon can then gently tighten the suture tension using the friction of the suture to hold the sclera in position, or a locking maneuver can be performed. This technique can be used for two or three sutures side by side, then the uveal can be systemically reposited as each suture is tightened. Additionally, the assistant (if available) can simply hold one end of the suture while the surgeon reposits the uvea and pulls the proximal end of the suture as the spatula is gently removed.

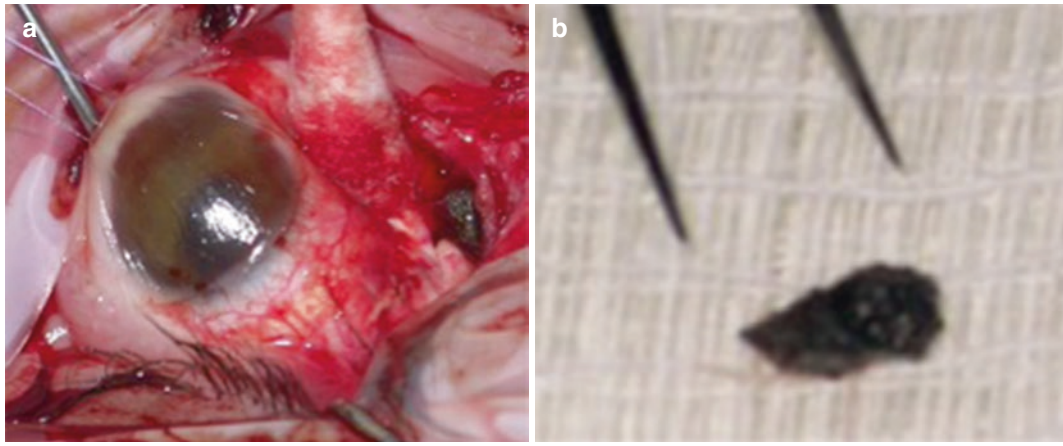

Fig. 6.9 (**a**) Orbital foreign body seen adjacent to the globe during a ruptured globe repair, masquerading as uvea. (**b**) The same foreign body seen after removal from the orbit

In globe ruptures from blast, or improvised explosive devices, it is not uncommon to find silica, wood, and rock injected through the cornea into the anterior chamber. Gentle vacuuming of the iris with the Simcoe I&A can remove the sand without damaging the iris. This should be attempted only if there is a clear view of the intraocular structures, as traction on unappreciated vitreous can cause additional trauma.

An additional phenomenon common to IED injuries not frequently noted in civilian trauma is the occurrence of multiple non-contiguous globe rupture sites. Thus, once the main wound is found and closed, a meticulous exploration of the globe is required in all cases, with particular attention to the area adjacent to the globe. Frequently large rocks and glass can be found adjacent to the globe. This is particularly true when one finds a comminuted scleral laceration, a wound frequently seen when glass enters the orbit and lacerates the sclera (Fig. 6.9a, b).

Once the ocular surface wounds are completed and the anterior chamber is free of debris, one has to decide the disposition of any lenticular damage. In penetrating injuries, it is not necessary to remove the lens at the initial operation, and can in fact be very risky. The combination of corneal edema, intraocular blood, and foreign bodies may make visualization difficult. It can be difficult to determine if a foreign body passed though the posterior capsule. Attempting to remove the lens in these circumstances can actually cause further injuries to the patient. Another consideration is that the lens capsule will fibrose over time, secondary to the intraocular inflammation. This process can facilitate delayed removal of lens material. Thus a planned staged procedure, after the pupil has been dilated, and there is vitreo-retinal surgery backup should strongly be considered.

Post Procedure

In mass casualty situations, one is not always able to communicate the extent of ocular injuries well with higher echelons of care. Whenever possible, one should include a detailed drawing of all injuries encountered in the medical notes, including suture types and locations and any concerns regarding the closure. In addition to verbal coordination, this will assist the subsequent ophthalmologist in their surgical planning for future operations. Immediately post procedure, the patient should receive subconjunctival antibiotics and steroids. The patient should be continued on prophylactic antibiotics and topical steroids in the immediate postoperative period. If there was bleeding in the anterior segment, one should consider the addition of a mydriatic medication like scopolamine or hom-

atroprine to relax the iris and ciliary body and minimize postoperative bleeding. Finally, the eye should be patched and protected from further injury.

References

1. Department of Defense, Joint Trauma Registry, Data Pull, Ocular Injuries 2003–2005, U.S. Army Institute of Surgical Research, Fort Sam Houston, Tx.
2. Cockerham GC, Rice TA, Hewes EH, Cockerham KP, Lemke S, Wang G, Lin RC, Glynn-Milley C, Zumhagen L. Closed-eye ocular injuries in the Iraq and Afghanistan wars. N Engl J Med. 2011;364(22):2172–3.
3. Colyer MH, Weber ED, Weichel ED, Dick JS, Bower KS, Ward TP, Haller JA. Delayed intraocular foreign body removal without endophalmitis during operations Iraqi freedom and enduring freedom. Ophthalmology. 2007;114(8):1439–47.
4. Pieramici DJ, MacCumber MW, Hunayum MU, Marsh MJ, De JJ. Open-globe injury, update on types on injuries and visual results. Ophthalmology. 1996;103(11):1798–803.
5. Russell SR, Olsen KR, Folk JC. Predictors of scleral rupture and the rule of vitrectomy in severe blunt ocular trauma. Am J Ophthalmol. 1988;105(3):253–7.
6. Klystra JA, Lamkin JC, Runyn DK. Clinical predictors of scleral rupture after blunt ocular trauma. Am J Ophthalmol. 1993;115(4):530–5.
7. Arras MH, Miloglu O, Barutcugil C, Kantaruci M, Ozcan E, Harorli A. Comparison of sensitivities of detecting foreign bodies among conventional radiography, computerized tomography and ultrasonography. Dentomaxillofac Radiol. 2010;39(2):72–8.
8. Weichel ED, Colyer MH, Ludlow SE, Bower KS, Eiseman AS. Combat ocular trauma visual outcomes during operations Iraqi and enduring freedom. Ophthalmology. 2008;115(12). Table 3 page 2238.
9. Weichel ED, Colyer M. Combat ocular trauma and systemic injury. Curr Opin Ophthalmol. 2008;19(6):P520.
10. Cho RI, Bakken HE, Reynolds ME, Schlifka BA, Powers DB. Concomitant cranial and ocular combat injuries during Operation Iraqi Freedom. J Trauma. 2009;67(3):516–20; discussion 519-520.
11. Current practice of thermoregulation in the transport of combat wounded. J Trauma. 2010;69(1):S162–7.
12. Michelson AD, MacGregor H, Barnard MR, Kestin AS, Rohrer MJ, Valeri CR. Hypothermia-induced reversible platelet dysfunction. Thromb Haemost. 1994;71:633–40.
13. Baruchin AM, Schaf S. Care of traumatic tattoos associated with gunpowder explosions and blast burns. In: Masellis M, Gunn S, editors. The management of mass burn casualties and fire disasters. Dordrecht: Springer; 1992. p. 292–3.
14. Vachon CA, Warner DO, Bacon DR. Succinylcholine and the open globe: tracing the teaching. Anesthesiology. 2003;99:220–3.
15. Macsai MS. Surgical management and rehabilitation of anterior segment trauma. In: Krachmer JH, Mannis MJ, Holland EJ, editors. Cornea. 2nd ed. St Louis: Elsevier-Mosby; 2005.
16. Eisner G. Eye surgery, an introduction to operative technique, 2nd fully revised and expanded edition. Berlin/Heidelberg: Springer. p. 94.
17. Macsai MS, Fontes BM. Trauma suturing techniques. In: Macsai MS, editor. Ophthalmic microsurgical suturing techniques. Berlin/Heidelberg: Springer; 2007. p. 65.
18. Eisner G. Eye surgery, an introduction to operative technique, 2nd fully revised and expanded edition. Berlin/Heidelberg: Springer. p. 91.
19. Benjamin L. The physics of wound closure, including tissue tactics. In: Macsai MS, editor. Ophthalmic microsurgical suturing techniques. Berlin/Heidelberg: Springer; 2007. p. 4.
20. Daoud YJ. The intraoperative impression of post operative outcomes of gamma irradiated corneas in cornea and Glaucoma patch surgery. Cornea. 2011;30(12):1387–91.
21. Macsai MS, Fontes BM. Trauma suturing techniques. In: Macsai MS, editor. Ophthalmic microsurgical suturing techniques. Berlin/Heidelberg: Springer; 2007. p. 67.
22. Bower KS. Sharp anterior segment trauma, USUHS ocular trauma course. Bethesda: Uniformed Services University of the Health Sciences; 2008. p. 13.
23. Eisner G. Eye surgery, an introduction to operative technique, 2nd fully revised and expanded edition. Berlin/Heidelberg: Springer; 2007. p. 188.
24. Akkin C, Kayikcioglu O, Erakgun T. A novel suture technique in stellate cornea lacerations. Ophthalmic Surg Lasers. 2001;32(5):436–7.

Posterior Segment

7

Marcus H. Colyer and Eric D. Weichel

Introduction

Injuries to the posterior segment are among the most complex to diagnose, challenging to effectively treat, and often become the defining injury to predict visual outcome. In order to effectively diagnose and treat these injuries, clear multidisciplinary communication and coordination are essential. Additionally, management of posterior segment injuries may require adaptation to traditional surgical approaches in order to achieve the optimal visual outcome.

The assessment of a casualty from military or civilian injuries begins with gathering relevant facts regarding the cause of the injury. Often, the ophthalmologist is one member of a team consisting of emergency physicians, trauma surgeons, orthopedic surgeons, critical care physicians, and other specialists who are consulted to manage the polytraumatic injuries (Fig. 7.1) [1]. Once consulted, the ophthalmologist's role is to accurately triage the ophthalmic injury, initiate initial treatment to stabilize the injury, and begin planning for definitive surgical intervention. Frequently, immediate posterior segment interventions are initially unnecessary but must be considered in the early days post injury in order to facilitate optimal visual outcome (Fig. 7.2) [2–5].

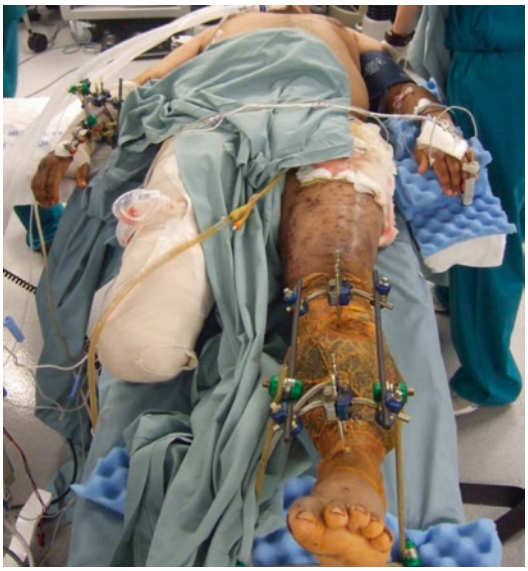

Fig. 7.1 Example of polytraumatic injury requiring extensive resuscitation measures, trauma surgery, orthopedic surgery, and ophthalmic head and neck team

The armamentarium to triage, diagnose, and treat includes critical equipment and honed diag-

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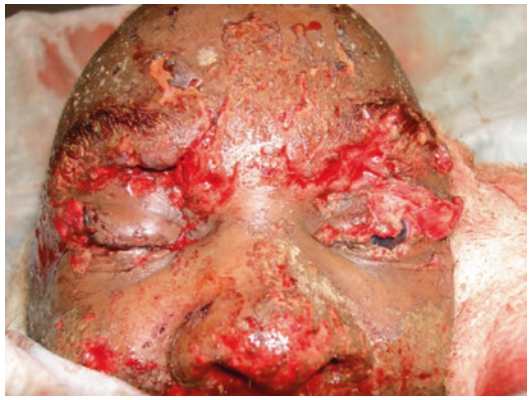

Fig. 7.2 Ocular polytrauma with eyelid laceration, open globe injury, and posterior segment trauma, requiring comprehensive evaluation and staged treatment to achieve optimal visual outcome

nostic skills. First, a thorough exam at the bedside should include visual acuity measurement, assessment of afferent pupillary defect, and assessment for the presence of signs of an open globe. If signs of open globe are present, the likelihood and severity of bacterial contamination must be assessed and emergent surgical intervention must be undertaken. There is no consensus regarding the precise timing of open globe closure, immediate closure is the standard, and the standard practice is to achieve primary globe repair within 24 hours, and otherwise it is a delayed closure [6]. Early antimicrobial prophylaxis is the standard of care and should consist of both topical and systemic antibiotics. Antibiotic coverage must include coverage for both gram-positive and gram-negative bacteria, given the relatively higher rate of gram-negative microbes in ocular trauma, and antimicrobials must achieve adequate intraocular penetration into the vitreous where a blood ocular barrier exists [7]. Diagnostic equipment for posterior segment injury includes indirect ophthalmoscopy, ophthalmic ultrasonography, and computed tomography. If signs of open globe injury are absent, accurate diagnosis, counseling, and discussion of treatment options with the patient, the primary team, and the patient's family are essential to maximize the potential for visual recovery (Fig. 7.2).

Closed Globe Injury

Closed globe injuries involving the posterior segment are often challenging to diagnose initially due to the presence of concomitant media opacity. In the presence of corneal edema, cataract, or vitreous hemorrhage, a careful history coupled with skilled diagnostic skills can result in early diagnosis of visually threatening conditions. Dilated ophthalmic examination with slit lamp biomicroscopy should occur as early as practical in the care of a combat casualty. Often in mass casualty situations, the exams are delayed hours or days due to systemic comorbidities [8]. When consulted, the exams may initially be limited due to patient unconsciousness or neurosurgical restrictions with regard to pupillary dilation. However, daily monitoring and attempts at examination with regular coordination with primary physicians will often result in a clear understanding of the treatment required to maximize visual outcome. If open globe injury can be excluded and media opacity is preventing view of the posterior segment, regular ophthalmic ultrasound with A- and B-scan capabilities will facilitate the anatomic disposition of the posterior segment and assist in determining surgical timing. Specifically, the presence of vitreous hemorrhage with traction, retinal tear, or retinal detachment can be discerned.

Vitreous Hemorrhage

Vitreous hemorrhage is a common finding associated with open and closed globe trauma, and the cause may be the result of intraocular hemorrhage that enters the vitreous cavity from any vascularized tissue (Fig. 7.3). When trauma is principally focused in the posterior segment, hemorrhage may be the result of avulsed retinal vessel, increased intracranial or intrathoracic pressure, shearing forces between the hyaloid face and retina, or may arise from more anterior structures such as the ciliary body, iris, or angle. Visual acuity at presentation can range from light perception to 20/20. Half of all vitreous hemorrhages related to closed globe ocular trauma are

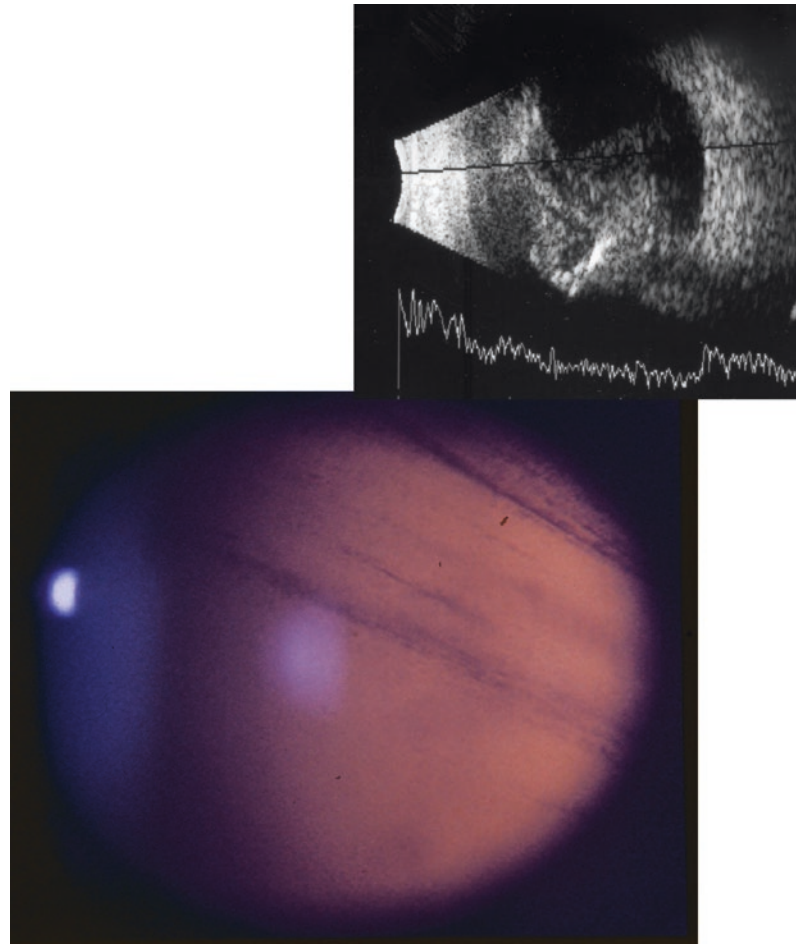

Fig. 7.3 Ophthalmic ultrasound and slit lamp findings associated with dense vitreous hemorrhage. Examination reveals absence of retinal detail with streaking of hemorrhage in vitreous while ultrasound reveals hyperechogenicity within vitreous cavity

associated with posterior segment pathology, so surgical intervention is often necessary [9]. However, with careful observation and regular examinations, vitreous hemorrhages can be monitored in the absence of retinal tear or detachment. If visual recovery is preventing physical or occupational rehabilitation, surgical intervention should be considered earlier even in the absence of retinal tear or detachment. Occasionally, long-term persistence of vitreous hemorrhage can result in red blood cells clogging the trabecular meshwork and result in ghost cell glaucoma.

Commotio Retinae

Commotio retinae was first described in 1873 by Berlin as a contusion of the retina resulting in extracellular edema and retinal whitening in a 12-year-old child [10]. It represents one of the classic examples of ocular damage resulting from trauma due to concussive changes within the retina. The pattern of trauma may result in compressive edema (contra coup) within defined zones of the retina—macula, midperipheral, or peripheral (Fig. 7.4). The constellation of findings and symptoms are variable and include mild visual distortion, photopsias, scotomas, vision loss, and lack of symptoms. If the contusion is located in the macula, it is more likely to result in vision loss and long-term visual dysfunction. Late findings include macular pigmentary mottling and/or pigment migration, while other cases are without findings. It is important to inspect the peripheral retina for changes consistent with commotio retinae even if visual acuity is normal.

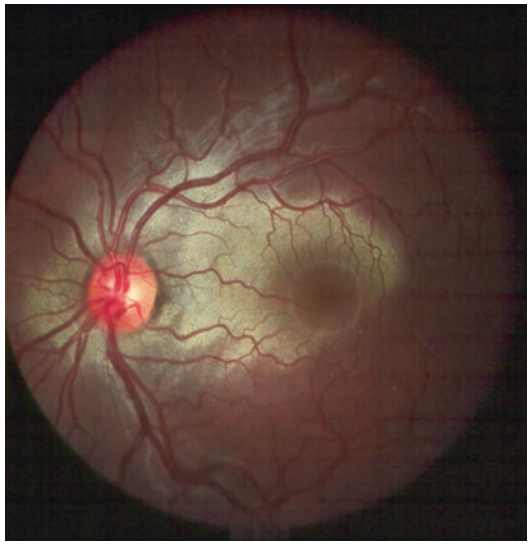

Fig. 7.4 Commotio retinae. Photo demonstrates retinal opacification in the macula as a result of closed globe "blunt" trauma to the left eye

Treatment of commotio retinae is conservative. Peripheral lesions resolve without consequence, while macular commotio changes often result in persistent visual field defects. Concomitant injuries must be evaluated and excluded, to include corneal endothelial dysfunction, angle recession, cataract, and zonular laxity following commotio injury.

Choroidal Rupture

Compressive injuries to the eye may result in crescentic tears of the inelastic Bruch's membrane, retinal pigment epithelium, and choriocapillaris as a result of direct closed globe ocular injuries [11]. Direct tears occur at the site of impact, while more commonly, the tears are located posteriorly in patterns concentric to the optic nerve of varying distances, but frequently may include the fovea and may result in permanent vision loss. Acutely, subretinal hemorrhage is evident and may obscure foveal-involving ruptures (Fig. 7.5). Typically, the hemorrhage clears within a few weeks with prominent gliosis and bare sclera forming at the site of the previous scar and hemorrhage. Choroidal neovascularization

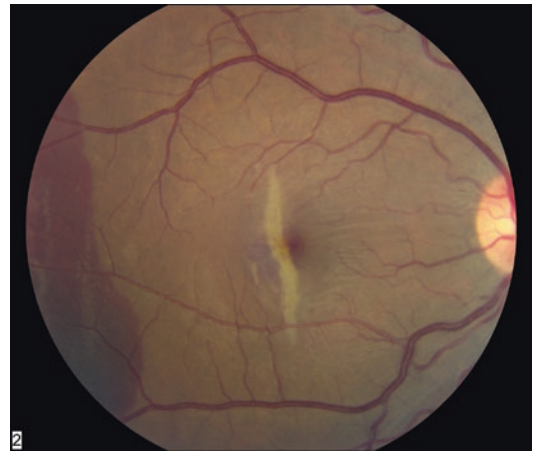

Fig. 7.5 Multiple choroidal ruptures. Peripheral macula reveals multiple choroidal ruptures involving the inelastic Bruch's membrane. Trauma associated with significant subretinal hemorrhage

can occur at any point following choroidal rupture, necessitating close observation in order to prevent late complications of neovascularization. Baseline visual acuity is often reduced regardless of the specific rupture site, while up to half of patients regain functional vision following choroidal rupture [12]. Fluorescein angiogram is helpful early in the post-injury course in order to visualize through hemorrhage and to identify early leakage from ruptured vessels. During the course of follow-up, optical coherence tomography (OCT) is critical to monitor for late effects of injury and assess the resolution of post-injury subretinal hemorrhage and fluid. Two OCT patterns are recognized with choroidal rupture—type 1 lesions are dome-shaped elevation of Bruch's membrane and RPE while type 2 lesions reveal a posterior bowing of Bruch's membrane and RPE. Type 2 lesions tend to predominate within the macula and may predispose to choroidal neovascularization later [13].

Macular Hole

Macular hole, as first described in 1975, is a well-known complication of closed globe ocular trauma and is the culmination of severe anteroposterior (A-P) propagation of forces following

direct trauma to the globe [14]. When the A-P forces reach the midpoint of the globe, equatorial expansion occurs, resulting in tangential traction in the macula. The unique vitreoretinal anatomy within the macula, to include overlying cortical vitreous, an adherent hyaloid face, and relatively thin foveal region predispose the fovea to dehiscence and subsequent hole formation.

Macular hole following closed globe injury spontaneously closes in many cases, although it is impossible to predict the likelihood of closure immediately post injury. Additionally, there may be concomitant macular injuries, including choroidal rupture, commotio retinae, and retinal pigment epitheliopathy as a result of direct retinal injury, and visual prognosis is highly variable [15].

If the hole does not spontaneously close within 4 weeks of injury, treatment for macular hole is surgical [4]. While monitoring the hole for a period of time extending up to a month post injury is reasonable, surgical intervention is frequently necessary. An isoexpansile concentration of intraocular gas of 3 to 6 weeks is standard, following a standard pars plana vitrectomy with internal limiting membrane peeling.

Retinitis Sclopeteria

Retinitis sclopeteria is classically referred to as a "claw-like" disruption of the choroid, retina, and overlying vitreous following a high-velocity projectile passing adjacent to the globe. Sclopeteric injuries are common following blast injuries due to the numerous small shrapnel objects that are released following blast. Sclopeteria injuries are prominent and peripheral but fortunately result in a low likelihood of retinal tear or detachment; rather, the injury results in firm chorioretinal adhesions and the authors have not found a case of rhegmatogenous retinal detachment associated with retinitis sclopeteria, although isolated hemorrhagic detachments involving the peripheral retina are common. Chorioretinal scarring is typically noted once hemorrhagic complications resolve (Fig. 7.6).

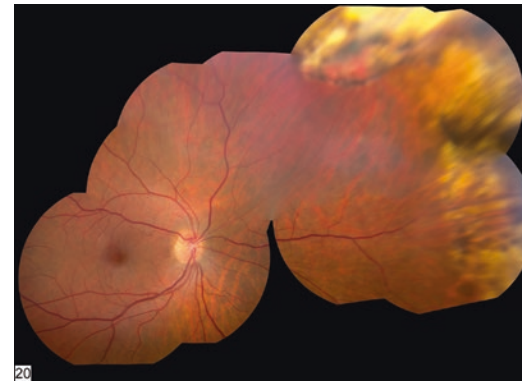

Fig. 7.6 Retinitis sclopeteria (remote). Hemorrhagic chorioretinal disruption in peripheral retina following significant ocular injury results in peripheral chorioretinal scarring but rarely, if ever, results in rhegmatogenous retinal detachment

Retinal Dialysis, Vitreous Base Avulsion, and Retinal Tear

Retinal dialysis forms as a congenital or acquired dehiscence of the retina at the ora serrata. The finding may be in any quadrant of the retina, although it is most commonly seen inferonasally. Retinal dialyses develop classically as a result of closed globe, blunt trauma, leading to equatorial expansion with a difference in the stretch characteristics of the retina and pars plana. Often, the dialysis is very subtle and requires careful scleral depression or visualization with contact biomicroscopy with angled lens. In the absence of accumulation of subretinal fluid, dialyses can be treated with laser retinopexy or cryopexy. Once subretinal fluid accumulates, scleral buckling is often necessary to close the dialysis and prevent vision loss.

A vitreous base avulsion is pathognomonic of closed globe ocular trauma. Often coined a "bucket handle" retinal dehiscence, the lesion involves both pars plana and retina. Specifically, the vitreous base straddles the ora serrata, extending 2 mm anteriorly from the ora serrata and 2 mm posteriorly extending just anterior to the equator with a wider base nasally [16]. With equatorial expansion of the globe following blunt trauma, the vitreous, tethered to the retina at the vitreous base, has a tendency to retract centrally

while the circumference of the globe increases, as with scleral stretching. The avulsion may be localized or extensive, and often includes a hemorrhagic component. Classically, the finding is noted when viewing the peripheral retina with the slit lamp or indirect ophthalmoscope with or without condensing lens. Treatment options include laser photocoagulation for small isolated avulsions, scleral buckling, if associated with peripheral fluid, and vitrectomy to remove the avulsed vitreous base.

Necrotic or trauma-associated retinal breaks occur in eyes with formed vitreous. Generally, there are firm attachment points between the vitreous and retina, either at the posterior vitreous base or areas of lattice degeneration or cystic retinal tufts. Following contusive ocular trauma, the potential space between the retina and vitreous may become manifest, resulting in increased traction on areas predisposed to tear formation. Posttraumatic retinal holes are often irregularly shaped, rather than the horseshoe-shaped retinal tears. Additionally, intraretinal hemorrhages are often clustered in areas of increased traction and signal to the examiner the potential presence of tiny holes. A high index of suspicion coupled with careful examination is critical to ensure retinal detachment is not missed. Treatment of necrotic breaks is generally the same as other retinal tears, although the presence of retinal and vitreous hemorrhage may make the treatment more difficult. Close observation while hemorrhage clears often allows for the complete barricade of the retinal tear.

Giant retinal tears are among the most challenging closed globe injuries to manage due to the propensity to result in retinal detachment. Giant retinal tear formation occurs similar to other contusive ocular injuries, though the tear in the retina tends to extend along the posterior vitreous base, resulting in at least 3 hours of retinal tear and tendency to rapidly collect subretinal fluid and scroll. If the tear is identified prior to retinal detachment, laser retinopexy is necessary, while the corners of the tear are the most critical areas to treat. However, if retinal detachment occurs, vitrectomy surgery is necessary to reattach the retina and prevent postoperative retinal slippage. During vitrectomy, careful vitreous base shaving, removal of the retinal traction, and relaxation of all traction on the edges of the tear are critical steps.

Open Globe Injury

The management of posterior segment complications of open globe injuries requires skill, ingenuity, and long-term postoperative monitoring to ensure optimal outcomes. Open globe injuries are classified as lacerations, ruptures, or mixed. Lacerations are the result of sharp injuries to the globe resulting in a full thickness of the cornea and/or sclera. When the object passes through the eye and exits the other side, it is considered a perforation. If the object remains within the eye, it is an intraocular foreign body, and if the object strikes the eye creating an wound, but does not remain in the eye, it is a laceration. Meanwhile, ruptures are the result of blunt force trauma which exceeds the sclera's ability to resist rupture and typically occurs in the thinnest areas of the sclera (Fig. 7.7).

Open globe injuries are classified according to the location of the most posterior extent of the ocular rupture or laceration. Zone 1 injuries are isolated to corneal lacerations/ruptures, zone 2 injuries extend from the limbus to 5 mm posterior to the limbus, and zone 3 injuries include all eye wall injuries posterior to zone 2 injury. Open globe injuries isolated to the anterior segment (zone 1 and some zone 2 injuries) do not necessarily require retinal surgical involvement. However, dilated funduscopic examination and/or B-scan ultrasonography are mandatory to exclude intraocular foreign body that may be hidden. Additionally, high-resolution orbital CT scan is always indicated in open globe injuries to ensure occult foreign bodies are not present. A primary repair of zone 1, 2, and 3 open globe injuries should be undertaken as soon as possible, with the standard of care being within 24 hours of injury (Fig. 7.8). Vitreoretinal surgical intervention is not required at the time of primary globe repair for any type of open globe injury and, in some cases, delayed vitreoretinal intervention

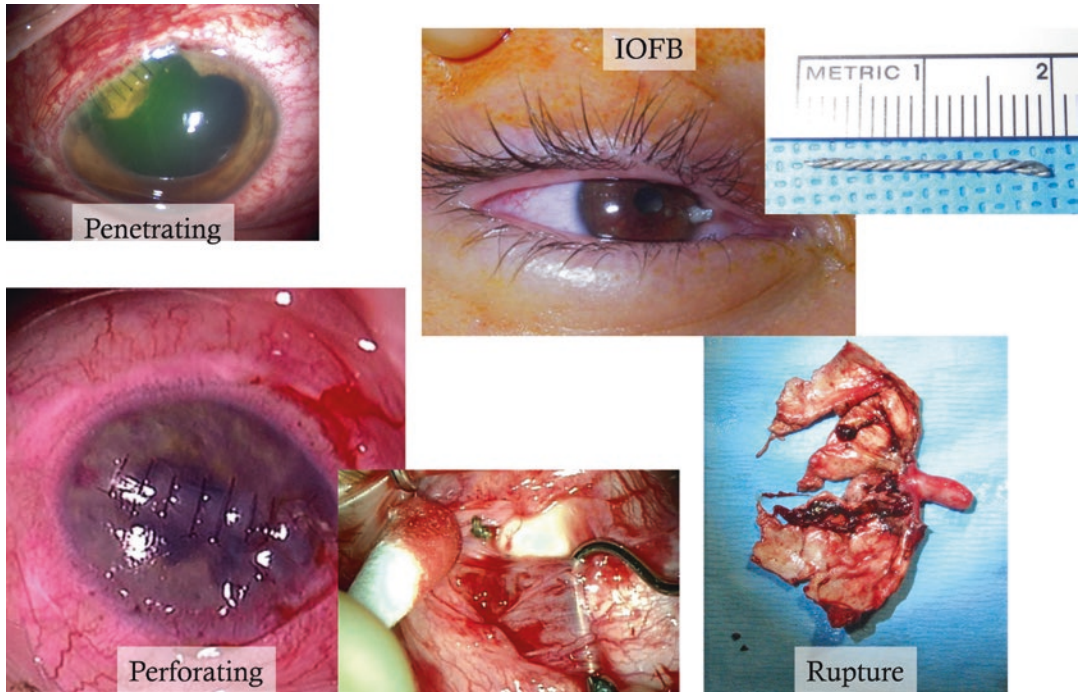

Fig. 7.7 Categories of open globe injuries. Representative examples of penetrating, IOFB, perforating, and rupture forms of open globe injury resulting in variable injury patterns and visual outcomes

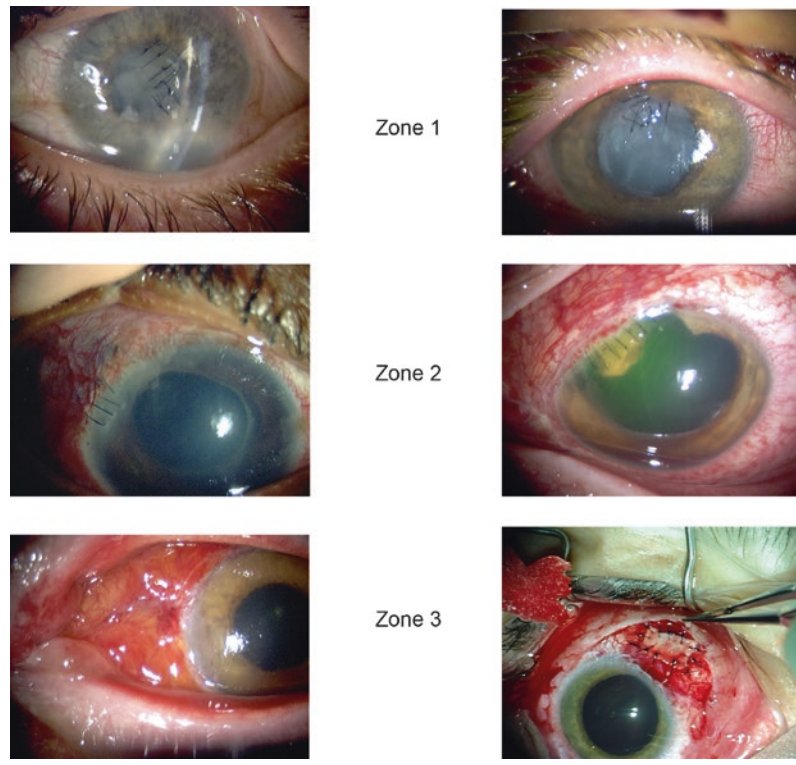

Fig. 7.8 Open globe injury. The injury noted must be evaluated and watertight closure must be achieved as soon as possible, regardless of intraocular pathology

may be desired. During the primary globe repair of zone 3 injuries, care must be taken to avoid uveal or retinal incarceration into the surgical wound, and indiscriminate debridement of the wound edges is not desirable.

Vitreoretinal Surgery

There are at least five vitreoretinal surgical indications for zone 1 and 2 open globe injuries following primary open globe repair: nonclearing vitreous hemorrhage, traumatic cataract with posterior capsular violation, retained posterior segment intraocular foreign body, traumatic rhegmatogenous retinal detachment, and posttraumatic endophthalmitis. When considering all of these situations, one must assume the visualization of the posterior segment could be variably impacted due to the presence of corneal injury and anterior chamber hemorrhage. Wide-angle vitreoretinal surgical visualization systems often assist the surgeon in clearing hemorrhage, locating/extracting intraocular foreign bodies, and repairing retinal detachments. Use of iris hooks may move the pupil away from a corneal opacity in order to successfully accomplish vitrectomy, and only a fraction of the total cornea needs to be clear cornea to successfully visualize posterior segment trauma via noncontact viewing systems [17]. When visualization remains difficult, endoscopic vitrectomy systems offer satisfactory visualization of posterior segment structures, although the learning curve is quite steep. The least desired visualization strategy is temporary keratoprosthesis with pars plana vitrectomy. This is due to the amount of surgical trauma induced by the combined vitrectomy and keratoplasty, high rate of graft failure postoperatively, and suboptimal long-term surgical outcomes [18]. When considering pars plana lensectomy, maintaining the anterior capsule will facilitate immediate or delayed placement of a sulcus lens and may also prevent anterior migration of silicone oil in cases of retinal detachment.

Posterior segment surgical intervention is typically required for all open zone 3 globe injuries. This is due to the high likelihood of retinal injury/traumatic retinal break with sharp wounds and the inside-out pattern of trauma resulting from open ruptures. Vitreoretinal surgery should not occur earlier than 5 days following zone 3 open globe injuries due to the need to maintain a watertight primary wound closure. If suprachoroidal hemorrhage is present, it is often necessary to wait at least 10 days prior to surgical intervention, while posterior vitreous detachment may occur in some eyes if intervention is delayed several weeks. When repairing zone 3 open globe injuries, complete vitrectomy (to include vitreous within the wound repair) is essential, and removal of as much hemorrhage as possible is best to minimize the risk of postoperative proliferative vitreoretinopathy (PVR). Zone 3 injuries often require silicone oil tamponade for a prolonged period, although small scleral defects that remain anterior to the equator may be successfully repaired with a medium- or long-term gas tamponade.

Intraocular Foreign Body Removal

An algorithm for the management of intraocular foreign bodies should be considered in preparation for removal (Fig. 7.9) [17]. When considering intraocular foreign body (IOFB) removal, IOFBs can be particularly challenging to (1) locate, (2) hold, and (3) extract from the eye. When attempting to locate foreign bodies, review of preoperative imaging studies will help localize the foreign body in the eye. Attention should be paid to foreign bodies that appear adjacent to the sclera, as they can occasionally bury within the subretinal space or suprachoroidal space, and extraction can be very difficult. Once in the eye, careful vitrectomy with scleral depression often uncovers foreign bodies that are hidden in the anterior vitreous along the pars plana inferiorly. Areas of hemorrhage within the retina serve as indicators of a retinal impact site and is a localizing sign. In some cases, foreign bodies can embed into the subretinal or suprachoroidal space, so a high index of suspicion in an area presumed to hold a foreign body will improve

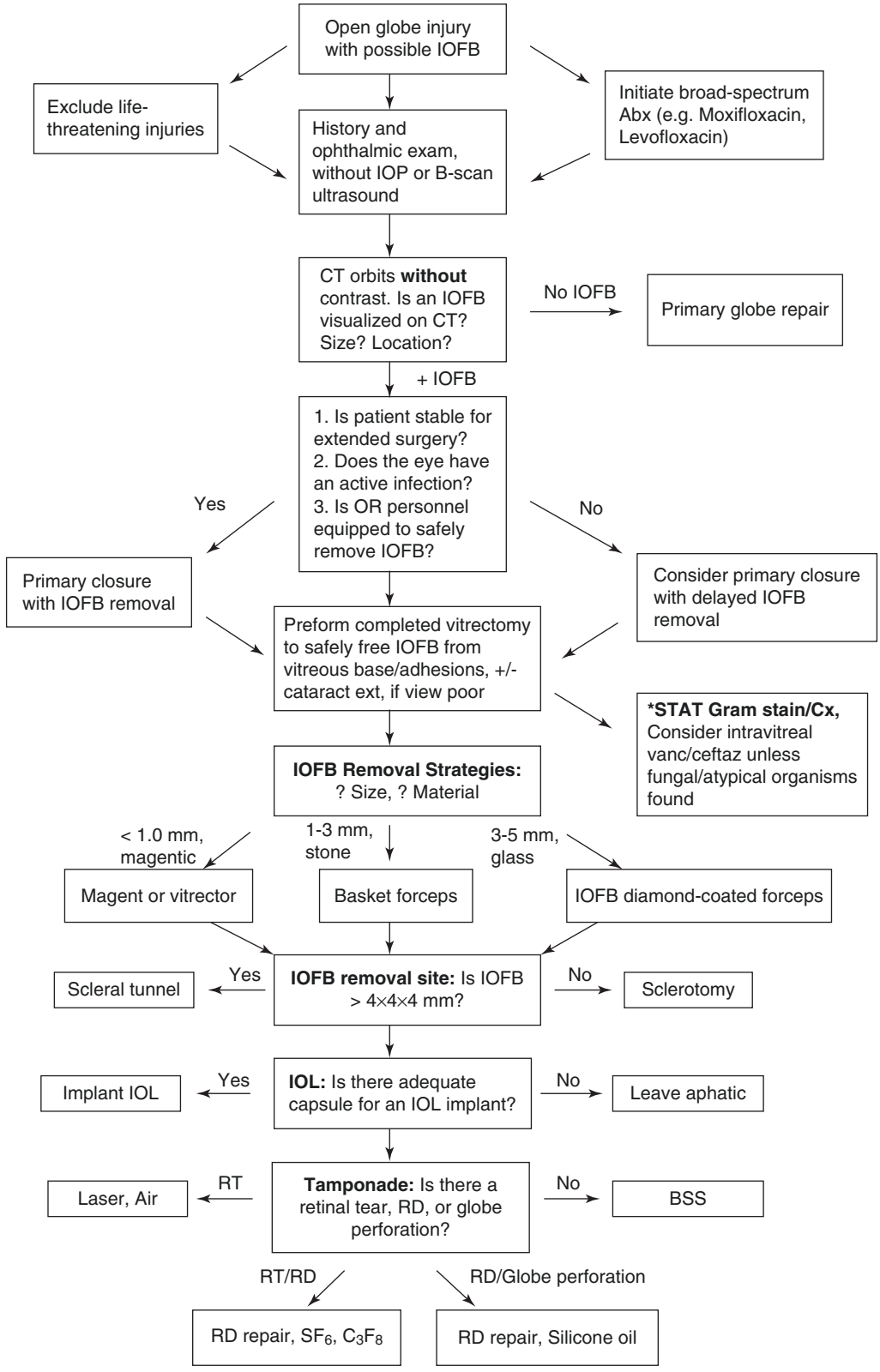

Fig. 7.9 Intraocular foreign body workflow. The algorithm listed provides a pathway for evaluating ocular injuries with intraocular foreign body injury as well as treatments based upon specific clinical parameters

surgical success, minimize intraoperative trauma, and generally lead to more favorable outcomes.

Once in surgery, careful planning of the best approach to the extraction site is necessary. Since posterior segment IOFB extraction is typically delayed from primary closure, the entrance site should not be used as the extraction site. Instead a clean surgical wound is necessary. With the transition to small-gauge vitrectomy surgery, sometimes tiny objects can be extracted through 25- or 23-gauge trocars. However, it is more likely that a sclerotomy will need to be enlarged to at least 20 gauge (1 mm) or up to 3 mm in order to extract IOFBs. Wounds larger than 3 mm are typically not advised within the sclera; instead, scleral tunnels with entry in the anterior segment will minimize postoperative astigmatism, scleral strength, and potential for retinal incarceration or iatrogenic retinal breaks at the sclerotomy site. However, scleral tunnels for IOFB extraction necessitate the removal of the crystalline lens, so in cases of large foreign body with a clear lens, the approach can vary.

Intraocular foreign body forceps are critical tools in the surgeon's armamentarium when considering foreign body removal (Fig. 7.10). Sometimes disposable 25- or 23-gauge trocars can be used for small foreign bodies. However, it is helpful to have accessibility to basket forceps, diamond-dusted grasping forceps, and an intraocular magnet (Fig. 7.11). Basket forceps are excellent to grasp and extract small and round foreign bodies. However, sharp edges can catch vitreous and/or retina when passing the foreign body through the sclera. Diamond-dusted forceps are particularly useful for extremely large foreign bodies and glass objects which tend to slip. Intraocular magnets hold ferrous IOFBs and minimize trauma at the wound. In all cases, a 0.12 style forceps will be necessary to stabilize the external portion of the IOFB during delivery of the object from the globe. Wounds should be enlarged beyond the size of

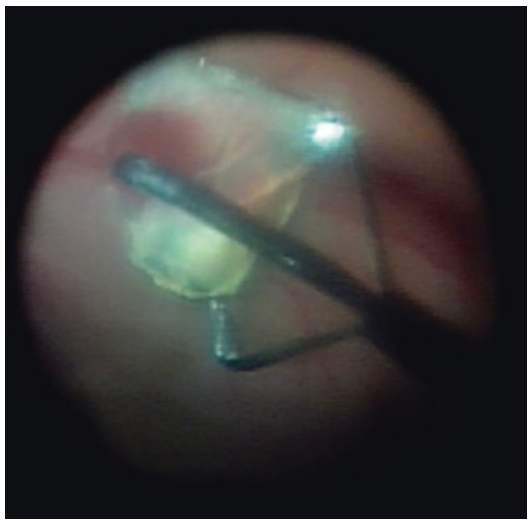

Fig. 7.10 Basket forceps. Grasping glass foreign body using a three fixation point basket forceps in order to stabilize and elevate a glass foreign body from the retinal surface in preparation for extraction

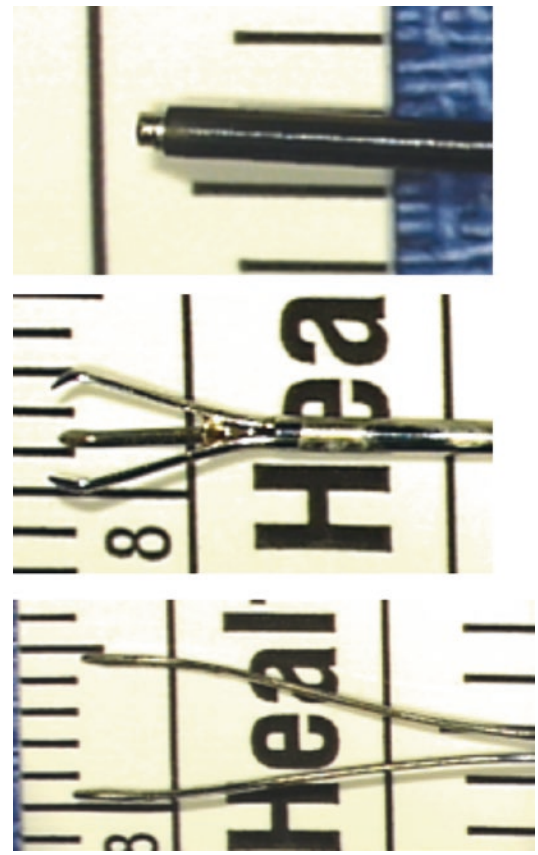

Fig. 7.11 Intraocular foreign body forceps. Example of an intraocular magnet, basket forceps, and positive action diamond-dusted forceps. The availability of a reliable set of intraocular foreign body forceps will ensure successful extraction of most foreign bodies encountered

the foreign body to ensure the object does not get stuck on the internal os of the sclerotomy. Some advocate the placement of cohesive viscoelastic or perfluorocarbon liquid over the macula to cushion against falling IOFBs in the macula.

Vitrectomy for Retinal Detachment

When considering vitrectomy in the presence of retinal detachment, the abovementioned factors must be considered and managed in concert with the retinal detachment. Once a detachment is noted, typically on ophthalmic ultrasound, surgical urgency is necessary to achieve an optimal visual outcome, though comorbidities and the medical status of the patient often contribute to surgical delays in comparison to standard rhegmatogenous retinal detachments. After considering the medical status of the patient, assessment of the patient's ability to position postoperatively, status of the cornea, and presence or absence of intraocular foreign bodies, the surgical plan can be considered. After a thorough vitrectomy with careful vitreous base shaving is accomplished, the internal aspect of scleral wounds should be inspected and attempts to eliminate as much vitreous as possible should be undertaken. Some have also advocated additionally removing any injured choroid and retinal pigment epithelium from the wound. If the laceration extends posterior to the equator of the globe, a scleral buckle could be considered to support the vitreous base and minimize potential for postoperative proliferative vitreoretinopathy. When considering reattachment approach, perfluorocarbon or internal drainage retinotomy approaches may be considered. Perfluorocarbon has the additional advantage of displacement of subretinal or choroidal hemorrhage away from the macula, but when corneal edema is present, the ability to remove all perfluorocarbon is a challenge. All tamponade agents can/should be considered. If intraocular gas is considered, communication with the primary team responsible for the patient is critical to prevent the use of nitrous oxide for several weeks. Silicone oil is often advantageous in cases of retinal detachment due to the permanent tamponade in these young and inflamed eyes.

Conclusion

Posterior segment injuries are highly variable in the visual impact, management, and surgical options. Counseling of the patient and family to understand the potential for poor outcome is critical from the outset, and they must understand that options may include numerous surgeries to achieve anatomic success. All open and closed globe injuries require evacuation for further evaluation as the recovery period is often weeks to months post injury. Ophthalmologists must be trained on critical skills such as to recognize the features of posterior segment injury, apply ophthalmic ultrasonography to accurately define the extent of pathology, and develop an appropriate evacuation strategy. Meanwhile, the skills of a vitreoretinal surgeon are challenged on every case of posterior segment trauma. No two injuries are alike while the surgical goal and desired outcome is—an attached retina with minimal macular injury or scarring with a functioning eye.

Disclaimer The opinions or assertions contained herein are the private ones of the author/speaker and are not to be construed as official or reflecting the views of the Department of Defense, the Uniformed Services University of the Health Sciences, or any other agency of the U.S. Government.

References

1. Weichel ED, Colyer MH. Combat ocular trauma and systemic injury. Curr Opin Ophthalmol. 2008;19(6):519–25.
2. Colyer MH, Weichel ED, Dick JS, Bower KS, Ward TP, Haller JA. Delayed intraocular foreign body removal without endophthamitis during Operations Iraqi Freedom and Enduring Freedom. Ophthalmology. 2007;114(8):1439–47.
3. Colyer MH, Chun DW, Bower KS, Dick JS, Weichel ED. Perforating globe injuries during operation Iraqi Freedom. Ophthalmology. 2008;115(11):2087–93.
4. Weichel ED, Colyer MH. Traumatic macular holes secondary to combat ocular trauma. Retina. 2009;29(3):349–54.

5. Weichel ED, Bower KS, Colyer MH. Chorioretinectomy for perforating or severe intraocular foreign body injuries. Graeves Arch Clin Exp Ophthalmol. 2010;248(3):319–30.
6. Thompson JT, Parver LM, Enger CL, Meiler WF, Liggett PE, National Eye Trauma System. Infectious endophthalmitis after penetrating injuries with retained intraocular foreign bodies. Ophthalmology. 1995;100(10):1468–74.
7. Bhagat N, Nagori S, Zarbin M. Post-traumatic infectious endophthalmitis. Surv Ophthalmol. 2011;56(3):238–9.
8. Yonekawa Y, Hacker HD, Lehman RE, Beal CJ, Veldman PB, Vyas NM, Shah AS, Wu D, Eliott D, Gardiner MF, Kuperwaser MC, Rosa RH, Ramsey JE, Miller JW, Mazzoli RA, Lawrence MG, Arroyo JG. Ocular blast injuries in mass-casualty incidents. Ophthalmology. 2014;121:1671.
9. Yeung L, Chen TL, Kuo YH, Chao AN, Wu WC, Chen KJ, Hwang YS, Chen YP, Lai CC. Severe vitreous hemorrhage associated with closed globe injury. Graefes Arch Clin Exp Ophthalmol. 2006;244:52–7.
10. Berlin R. Sogenannten commotio retinae. So-called commotio retinae. Klin Monatsbl Augenheilkd. 1873;1:42–78.
11. Von Graefe A. Ze Falle von Rupture der Choroida. Graefes Arch Clin Exp Ophthalmol. 1854;1:402.
12. Ament CS, Zacks DN, Lane AM, Krzystolik M, D'Amico DJ, Mukai S, Young LH, Loewenstein J, Arroyo J, Miller JW. Predictors of visual outcome and choroidal neovascular membrane formation after traumatic choroidal rupture. Arch Ophthalmol (Chicago, Ill: 1960). 2006;124(7):957–66.
13. Nair U, Soman M, Ganekal S, Batmanabane V, Nair K. Morphological patterns of indirect choroidal rupture on spectral domain optical coherence tomography. Clin Ophthalmol (Auckland, NZ). 2013;7:1503.
14. Weinstock SJ, Morin JD. Traumatic macular hole. Can J Opthalmol. 1976;11:249–51.
15. Williams DF, Mieler WF, Williams GA. Posterior segment manifestations of ocular trauma. Retina. 1990;10(Suppl):S35–44.
16. Ahmad B, Shah G, Engelbrecht N, Thomas M, Smith B. Chapter 24: Retinal detachment repair: scleral buckling procedures. In: Ichhpujani P, Spaeth G, Yanoff M, editors. Expert techniques in ophthalmic surgery. 1st ed. Philadelphia: Jaypee Hights Medical Pub Inc; 2015. p. 224.
17. Yeh S, Colyer MH, Weichel ED. Current trends in the management of intraocular foreign bodies. Current Opin Ophthalmol. 2008;19(3):225–33.
18. Chun DW, Colyer MH, Wroblewski KJ. Visual and anatomic outcomes of vitrectomy with temporary keratoprosthesis or endoscopy in ocular trauma with opaque cornea. Ophthalmic Surg Lasers Imaging. 2012;43(4):302–10.

Traumatic Glaucoma

8

Won I. Kim

Introduction

Glaucoma secondary to traumatic injury is a potentially devastating complication that may present acutely or develop many years later [1]. The 6-month incidence of developing post-traumatic glaucoma can be as high as 3.4% after blunt trauma and 2.7% after penetrating trauma [2, 3]. Thorough evaluation as well as vigilant and careful follow-up is necessary to detect and initiate therapy to prevent glaucoma from causing permanent and irreversible vision loss.

Glaucoma, in its purest definition, is an optic neuropathy characterized by a particular pattern of optic nerve neuroretinal rim changes and retinal nerve fiber layer loss that ultimately results in a characteristic pattern of visual field loss. Intraocular pressure (IOP) is just one of many factors that play a role in the pathogenesis of glaucoma, but it remains the only modifiable risk factor and so the management of glaucoma revolves around lowering it. The higher the IOP, the greater the risk of developing glaucoma, but not all patients with elevated IOP will develop glaucoma and certainly not all patients with glaucoma have elevated IOP.

But in the context of ocular trauma, not all patients will present with actual identifiable glaucomatous optic neuropathy meeting the definition of glaucoma, but they will most assuredly present with elevated IOP that presents a high risk of causing glaucomatous optic neuropathy if it is not dealt with in a timely fashion. So, for the purposes of this discussion, the term *glaucoma* will be used in a broad sense to describe conditions associated with trauma that result in high IOP that if not lowered sufficiently will be presumed to cause eventual optic neuropathy.

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Glaucoma Related to Non-penetrating Trauma

Glaucomas Secondary to Blood

Hyphema

Hyphema is the most common complication of the anterior segment after trauma. The violent compression of the globe caused by blunt trauma results in a distortion of the highly vascularized ciliary body and iris which can result in bleeding. The bleeding eventually stops, secondary to the elevated intraocular pressure and clot formation. The clot reaches maximum organization in approximately 4–7 days. The clot then undergoes fibrinolysis and the blood and breakdown products are subsequently removed from the anterior chamber via the trabecular meshwork.

Documenting the extent of the hyphema within the anterior chamber is an important part

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of the initial evaluation. A helpful method is to grade the amount of blood within the anterior chamber. Grade I is less than one-third filling of the anterior chamber, grade II is one-third to one-half, grade III is one-half to near total filling, and grade IV hyphema is a complete filling of the anterior chamber (Fig. 8.1) [4]. This is important as the prevalence of elevated IOP is related to the amount of hyphema. In one study of 235 cases, eyes with grade I–II hyphemas had only 13.5% develop elevated IOP, whereas in grade IV hyphemas 52% had elevated IOP [5]. A total hyphema with bright red blood should be distinguished from an *eight-ball hyphema*, which is characterized by dark reddish black blood. An eight-ball hyphema is a clot that is black because of decreased oxygen concentration. The resultant impaired aqueous circulation in the anterior chamber can lead to pupillary block and secondary angle-closure glaucoma.

Potential complications associated with hyphema include elevated IOP, corneal blood staining, uveitis, a vitreous hemorrhage from spillover of red blood cells from the anterior chamber, or rebleeding into the anterior chamber [4–6]. Rebleeding can occur as the initial clot begins to retract and lyse from day 2 to day 5. Prevalence varies from 3.5% to 38% [7]. Risk factors for rebleeding include: those with ocular hypotony or elevated IOP [8], a 50% or greater hyphema [9], systemic hypertension [10], use of aspirin [11], and black patients [12]. Rebleeds are significant in that they tend to be more severe than the initial bleed and have a higher likelihood of leading to complications such as elevated IOP, corneal blood staining, and synechiae formation (Fig. 8.2) [4, 7]. The conversion of plasminogen to plasmin by plasminogen activator promotes fibrinolysis. This process can be inhibited with anti-fibrinolytics, like aminocaproic acid, which has been shown in some studies to reduce the risk of rebleeding [13]. Side effects include postural hypotension, nausea, and vomiting. Tranexamic acid is a more potent anti-fibrinolytic than aminocaproic acid and may have fewer side effects [14, 15]. Of note, patients with polytrauma may already be receiving tranexamic acid for systemic hemorrhage control. Conversely, victims of severe polytrauma with hyphema may remain hospitalized and intubated for weeks on Lovenox for deep vein thrombosis prophylaxis which may then elevate the risk for rebleeding. These patients will need to be followed closely with daily monitoring until the hyphema clears.

The vast majority of hyphemas can be managed in an outpatient setting with precautions given to the patient to limit ambulation and activity and to wear eye protection. Hospitalization can be considered for those with very large hyphemas, sickle cell disease or trait, and unreliable patients who will not be able to follow instructions. The use of aspirin and nonsteroidal anti-inflammatory drugs should be avoided. Topical corticosteroids should be used to decrease

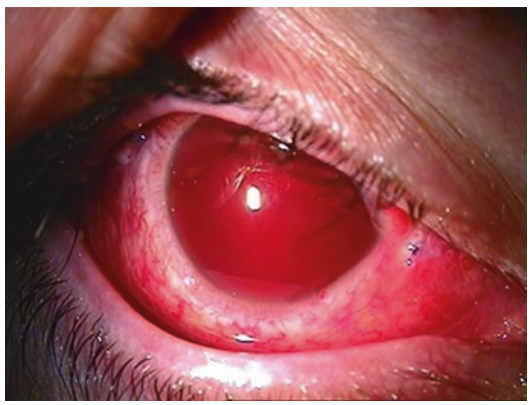

Fig. 8.1 Total hyphema

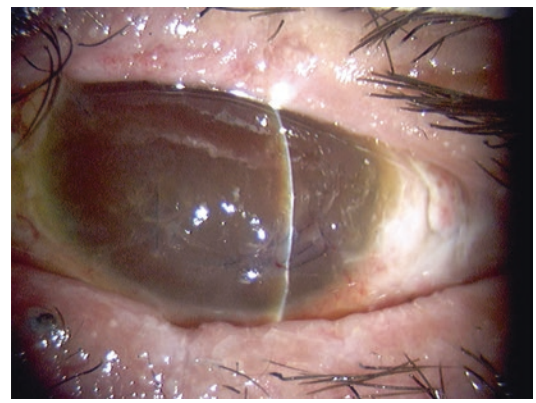

Fig. 8.2 Hyphema with rebleeding and corneal blood staining

inflammation. Topical cycloplegics should be used to prevent iris movement and subsequent clot dislodgement to reduce the risk of rebleeding and to prevent the formation of posterior synechiae.

Elevated IOP caused by hyphemas will typically be treated first with topical aqueous suppressants such as beta adrenergic antagonists, carbonic anhydrase inhibitors, and alpha adrenergic agonists. If topical agents are insufficient, systemic acetazolamide can be administered. Hyperosmotic agents such as mannitol can be considered if aqueous suppressants prove insufficient. Miotics and prostaglandin analogues are likely of little benefit in this scenario.

Sickle cell anemia is an important consideration in hyphema management. In this condition, a slight decrease in oxygen concentration can cause red blood cells to sickle, becoming deformed and rigid. These sickled cells can obstruct the trabecular meshwork and impede outflow of aqueous and elevate IOP dramatically at times vastly out of proportion to the amount of blood in the anterior chamber. Sickle cell is more common in the black population affecting approximately 10% but is also found in Hispanic patients. A history should be elicited and sickle cell prep should be sent on all patients who may be at risk. It is also important to note that even those with just sickle trait are at similar risk for these complications. In one report, 13 of 99 children with hyphema had sickle cell trait. Eleven of the thirteen sickle cell patients had elevated intraocular pressure and six required anterior chamber washout to control the intraocular pressure [16]. In addition to the risk of elevated IOP, sickle cell patients are also at increased risk for devastating vaso-occlusive events such as central retinal artery occlusion and ischemic optic neuropathy due to intravascular sickling even at mildly elevated IOP [4]. It is therefore prudent to consider early surgical intervention if the IOP is not controlled in the first 24 hours in this setting. Certain medications should be avoided in sickle cell patients. Topical carbonic anhydrase inhibitors (CAI) can increase ascorbic acid in aqueous humor and promote sickling [17]. Adrenergic agonists with alpha-1 activity such as Iopidine can promote intravascular sickling by their vasoconstrictive and subsequent deoxygenating properties. Hyperosmotic agents and systemic CAI have a risk of inducing sickle crises especially if the patient is dehydrated.

Surgical intervention is required in about 5% of hyphema cases. Surgical management should be considered if the IOP is too high despite maximum tolerable medical therapy and is deemed a threat to the optic nerve, if there is corneal blood staining, or if there is a total hyphema for more than 4 days (Fig. 8.3) [18]. Thresholds for recommending surgery for elevated IOP are the following: IOP greater than 50 mmHg for 5 days, IOP greater than 35 mmHg for 7 days, or greater than 24 mmHg for 24 hours in those with sickle cell or trait [19]. Surgical management usually consists of anterior chamber washout of the hyphema by either simple irrigation with balanced salt solution through a paracentesis or by the use of an irrigation and aspiration handpiece. Large clots, however, may best be managed by an anterior vitrectomy system. An added advantage of an anterior vitrector is the ability to debulk the clot in small amounts without shearing or violent fluid shifts that may cause renewed bleeding. Care must be taken especially in the setting of total hyphemas and eight-ball hyphemas to be cognizant of which direction the cutting port of the vitrectomy handpiece is facing and where the tip is inside the anterior chamber to ensure that no damage is caused to the endothelium, the iris, or the lens capsule. Anterior chamber washout alone

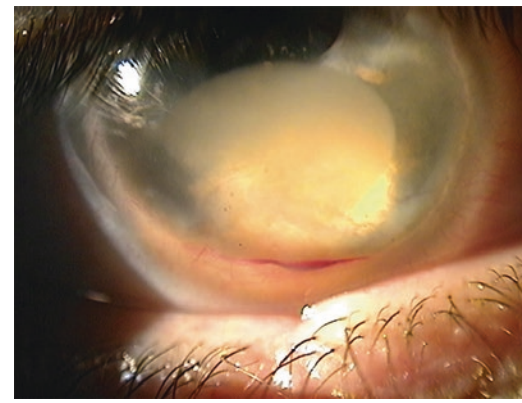

Fig. 8.3 Corneal blood staining

is usually sufficient to lower IOP without additional filtering procedures [20], but some have championed the use of trabeculectomy combined with anterior chamber washout especially in the setting of those with preexisting glaucomatous optic neuropathy, sickle cell disease, and total or eight ball hyphemas [18]. Performing this combined technique addresses all potential issues, it clears the blood, it lowers the IOP, and the iridectomy performed with the trabeculectomy will break any existing pupillary block. In this setting, it may be prudent to perform the trabeculectomy without the adjunctive use of mitomycin C as the long-term survival of the filtering bleb is not as important as in the usual setting of chronic glaucoma.

Once the blood in the anterior chamber has completely cleared, the job of the ophthalmologist is not complete. Angle recession is common in eyes that have sustained a blunt injury severe enough to cause a hyphema. All patients should be evaluated with gonioscopy once their hyphema has cleared to look for any signs of angle damage ranging from subtle tears in the trabecular meshwork to frank angle recession. If such pathology is discovered, that patient must be monitored for the rest of his or her life to look for the development of elevated intraocular pressures and glaucomatous optic neuropathy. Angle recession is discussed in greater detail later in the chapter.

Hemolytic Glaucoma

Hemolytic glaucoma occurs when hemoglobin-laden macrophages obstruct the trabecular meshwork and obstruct aqueous outflow. This usually develops days to weeks after a large intraocular hemorrhage. The anterior chamber may be filled with red-tinged cells and gonioscopy will reveal reddish brown pigmented deposits on the inferior trabecular meshwork. Most cases respond well to medical management and are self-limited. Rarely anterior chamber washout and/or vitrectomy may be necessary [4].

Ghost Cell Glaucoma

An old vitreous hemorrhage present for several months in an eye with a disrupted anterior hyaloid face can develop elevated IOP if degenerated red blood cells enter the anterior chamber and obstruct the trabecular meshwork. The diagnosis is made when khaki-colored cells are noted in the anterior chamber. These cells are called "ghost cells" for their appearance on histopathology where the degenerated erythrocytes are mostly, empty except for the presence of small clumps of denatured hemoglobin called "Heinz bodies." Ghost cells are especially adept at obstructing the trabecular meshwork because they are rigid and not deformable in the way a typical normal red blood cell is. Clues that distinguish this entity from uveitis are the lack of aqueous flare and the lack of conjunctival hyperemia relative to the cellular reaction seen. Ghost cell glaucoma can usually be managed with medical therapy and is usually a transient phenomenon but may take months for it to resolve. Uncommonly, refractory cases may require an anterior chamber washout and pars plana vitrectomy [4].

Hemosiderotic Glaucoma

Hemosiderotic glaucoma is also caused by a long-standing vitreous hemorrhage when free hemoglobin from lysed red blood cells are ingested by the trabecular meshwork endothelial cells. Iron from hemoglobin induces siderosis and the toxic effect on the trabecular meshwork cells, alters their function, and elevates IOP [4].

Angle Recession Glaucoma

Angle recession is a particular marker of injury to the anterior segment that is associated with the development of elevated IOP and resultant glaucomatous optic neuropathy. It is defined histologically as a separation between the longitudinal and circular muscle fibers of the ciliary body. Clinically it is diagnosed with gonioscopy with the classic finding of a widened ciliary body band (Fig. 8.4). In subtle cases, this widening effect may only be noticeable as a potential pathology when comparing it to other portions of the angle that are uninjured in the same eye or by

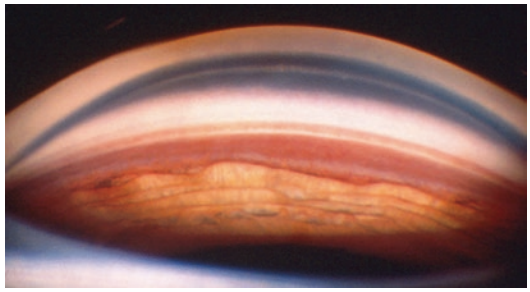

Fig. 8.4 Angle recession on gonioscopy showing an abnormally wide ciliary body band

comparison to the contralateral non-traumatized eye. Other gonioscopic findings associated with traumatic angle damage include sectoral areas of broken iris processes and an unusually prominent-appearing scleral spur.

Angle recession is a common finding in eyes with a history of traumatic hyphema, occurring in 60–94% of such eyes [21–24]. The presence of hyphema should automatically trigger an evaluation with gonioscopy to look for angle recession as soon as the blood clears. However, gonioscopic evaluation should also be undertaken for any eye that has suffered significant blunt trauma even in the absence of hyphema to ensure that angle recession is not missed. Missing such a diagnosis could lead to tragic vision loss and blindness as open angle glaucoma is a painless and asymptomatic disease. For example, a patient being followed for commotio retinae and/or vitreous hemorrhage after trauma should be evaluated with gonioscopy as the same blunt trauma that caused the posterior segment findings could have also injured the anterior segment. Likewise, a patient being followed for corneal contusion or corneal foreign bodies following a blast injury may also have suffered significant blunt trauma to cause angle recession and thus should be evaluated with gonioscopy when possible.

Four to nine percent of patients with angle recession will develop elevated IOP and glaucoma. Curiously, the IOP elevation associated with angle recession does not usually occur in the immediate post-injury period. Indeed, many times the IOP elevation and subsequent glaucoma occur many years later [25]. Angle recession is not directly the cause of elevated IOP, it is just a readily identifiable marker of injury to the angle structures that includes injury to the trabecular meshwork that may be occult and not readily observable. Wolff and Zimmerman were the first to suggest that degenerative changes in the trabecular meshwork tissue were the likely cause of elevated IOP after trauma [26]. Herschler supported this theory demonstrating in animal studies that tears in the trabecular meshwork could be seen in the early post-traumatic period. These tears heal and the subsequent scarring of the trabecular meshwork leads to increased resistance to aqueous outflow and elevated IOP [27].

Patients with angle recession are predisposed to be steroid responders and to developing primary open-angle glaucoma [28]. Those who develop angle recession glaucoma have up to a 50% chance of developing primary open-angle glaucoma in the contralateral non-traumatized eye [29]. It is likely that many of those who develop angle recession glaucoma have an underlying predisposition to develop primary open-angle glaucoma and the trauma to the angle just accelerates the process and hastens its onset. Angle recession glaucoma is treated similarly to other open-angle glaucomas. However, patients with angle recession have been noted to respond very poorly to laser trabeculoplasty. In one small series of 13 patients, there was only a 23% success rate [30]. Angle recession and traumatic glaucoma patients have an increased risk for trabeculectomy failure, so glaucoma drainage implants may be a more appropriate choice in this setting [31, 32]. All the various commercially available glaucoma drainage implants, the Molteno (IOP, Inc., Costa Mesa, CA, USA, and Molteno Ophthalmic Limited, Dunedin, New Zealand), the Ahmed Glaucoma Valve (New World Medical, Rancho Cucamonga, CA, USA), and the Baerveldt Glaucoma Implants (Advanced Medical Optics, Inc., Santa Ana, CA, USA), have all shown efficacy at lowering IOP in angle recession patients [33].

Other Causes of Post-Traumatic Glaucoma

Glaucomas Related to Lens Trauma

Pupillary Block Angle Closure Secondary to Lens Subluxation

Trauma can cause zonular disruption which can lead to crystalline lens subluxation. If the subluxed lens moves anteriorly it can cause a secondary angle closure due to pupillary block. If this is suspected, cycloplegics should be administered to tighten the remaining zonules which should flatten the overall curvature of the lens and draw it more posterior. The patient should be placed in a supine position to assist in repositing the lens in the posterior chamber. If this maneuver is successful and pupillary block is broken, a miotic should be administered and two laser peripheral iridectomies should be placed 180° apart. Some patients may require lens extraction by extracapsular or intracapsular means depending on the degree of zonular damage. In extreme cases where there is lenticular corneal contact, urgent surgical intervention will be required [4, 34].

Lens Particle Glaucoma

In trauma resulting in lens capsule disruption, free lens particles can cause elevated IOP by several different mechanisms. Lens particles themselves can obstruct the trabecular meshwork. Alternatively, the lens material can induce an inflammatory response which can lead to uveitic mechanisms of IOP elevation. Posterior synechiae can cause pupillary block and peripheral anterior synechiae can lead to a secondary angle closure. The management includes medical therapy to control the IOP, cycloplegics to prevent posterior synechiae, and topical corticosteroids to control the inflammation. If the elevated IOP cannot be controlled medically, surgical removal of the lens material may be necessary [4, 34].

Aphakia and Pseudophakia

Patients who have undergone lensectomy and vitrectomy for significant lens trauma and who have traumatic mydriasis may develop angle closure (Fig. 8.5). The lack of posterior pulling of the lens-zonule complex and the lack of tone in the iris and iris sphincter can gradually lead to total circumferential irido-trabecular adhesion in a process similar to the angle closure that can occur

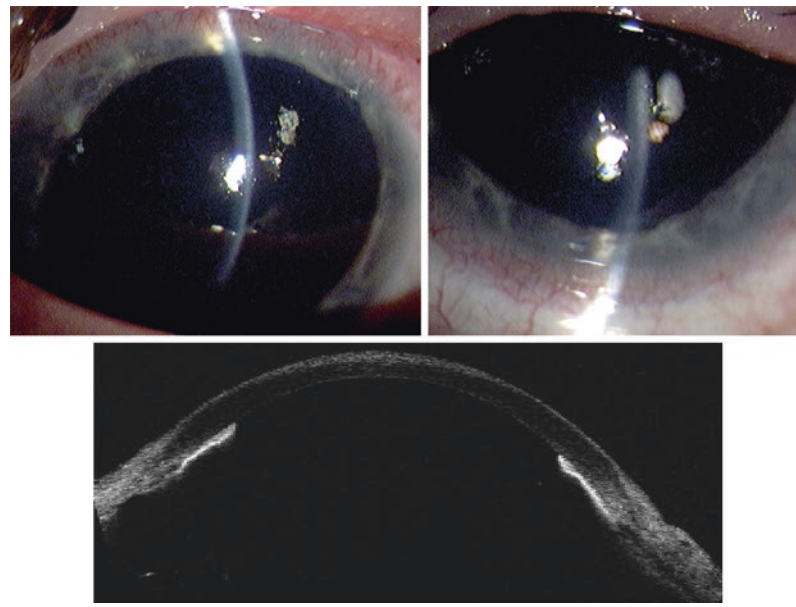

Fig. 8.5 Angle closure in an aphakic eye seen with slit lamp biomicroscopy and on anterior segment optical coherent tomography

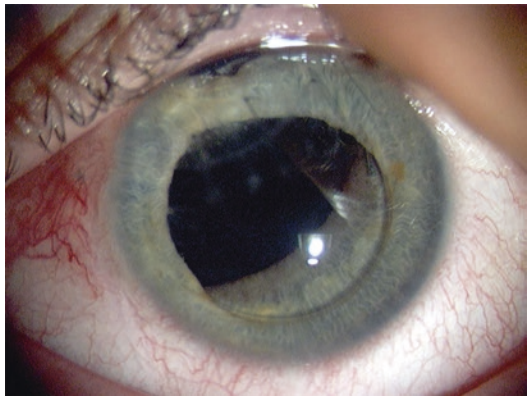

Fig. 8.6 Subluxed posterior chamber intraocular lens

in aniridia. Artificial intraocular lenses can also displace or sublux after trauma and cause glaucoma by pupillary block or by a uveitic or hyphema mechanism (Fig. 8.6).

Uveitis, Posterior Segment and Orbital Mechanisms

Uveitis

Uveitis associated with trauma can cause elevated IOP by several different mechanisms. Direct inflammation of the trabecular meshwork, trabeculitis, can cause elevated IOP as can the accumulation of inflammatory precipitates in the angle obstructing aqueous outflow. Chronic, persistent inflammation can cause posterior synechiae leading to pupillary block angle closure or anterior synechiae leading to chronic angle closure. Prolonged use of corticosteroids used to treat inflammation can elevate IOP as well.

Posterior Segment

Uveal effusion occurring after trauma can induce angle closure by a posterior pushing mechanism [35]. Vitreous prolapse into the anterior chamber has been reported to cause elevated IOP [36]. Rhegmatogenous retinal detachment after trauma can lead to Schwartz-Matsuo syndrome, where free floating photoreceptor outer segments obstruct the trabecular meshwork and cause IOP elevation [37].

Orbit

Orbital hemorrhage from trauma can cause a compartment syndrome which can elevate IOP and requires an urgent canthotomy and cantholysis. A carotid cavernous sinus fistula secondary to head trauma can cause elevated episcleral venous pressure which can result in elevated IOP.

Glaucoma Related to Penetrating Trauma

IOP after penetrating ocular injury is usually low because of the open wound. After proper surgical repair, the IOP can elevate due to several different mechanisms. Early in the postoperative course, hyphema and inflammation can potentially elevate IOP. The inflammation can lead to posterior synechiae causing pupillary block angle closure or peripheral anterior synechiae resulting in chronic angle closure. Corticosteroids and cycloplegics can be utilized to suppress the inflammation and prevent synechiae formation.

Epithelial downgrowth is a potentially devastating complication typically occurring from an insufficiently closed wound. The epithelial tissue will migrate over the angle and other anterior segment structures obstructing aqueous outflow. A gray or translucent membrane with a thickened, scalloped leading edge can be seen growing along the posterior cornea often accompanied by overlying edema of the corneal stroma. If the membrane is growing over the iris, it will give it a flattened, smooth, and cryptless appearance. To confirm the diagnosis argon or diode laser can be applied to a portion of affected iris and characteristic white burns of the epithelial tissue will appear. Aqueous suppressants, glaucoma drainage implants, cyclodestruction, and other heroic measures can be employed, but the prognosis is poor.

Retained intraocular foreign bodies (IOFBs) containing iron can cause siderosis, and the resulting toxicity to the trabecular meshwork can lead to a secondary open-angle glaucoma. An IOFB containing copper can also cause a similar reaction due to chalcosis but is typically not as severe [4, 34].

Glaucoma Related to Chemical Burns

Chemical burns can cause severe damage to ocular tissues. Alkali injuries are potentially more dangerous than acid injuries. Acid denatures proteins and this can serve as a barrier to further penetration into the eye. Alkali, however, can often penetrate directly into the anterior chamber within seconds of contact. Elevated IOP occurring immediately after chemical exposure can be due to scleral shrinkage and release of prostaglandins [38]. Elevated IOP in the intermediate period is often due to inflammation. Late IOP elevation after chemical injury is usually due to irreversible trabecular damage and/or peripheral anterior synechiae formation as a late sequela of inflammation.

Measurement of IOP with typical Goldmann applanation tonometry may be difficult in the setting of chemical injury due to severely disorganized and irregular corneas. A Tonopen applied to a relatively normal part of the cornea may be a good alternative for IOP measurement that is easily accessible in most situations. If one is available, a pneumatonometer (Model 30, Reichert, Depew, NY) would be an ideal way to measure IOP in this scenario, as it is very accurate even in the setting of a severely disorganized cornea. It is uncertain how newer modalities such as the Rebound tonometer (iCare, Vantaa, Finland) and the Diaton transpalpebral tonometer (BiCOM Inc., Long Beach, NY), which takes the IOP measurement on the superior sclera through the upper eyelid, would perform in the setting of severe chemical injuries.

Topical ophthalmic medications with their preservatives may inhibit reepithelialization of the ocular surface after chemical injuries. It may be prudent when possible then to consider the use of oral or IV agents such as acetazolamide and methazolamide, or preservative-free medications such as preservative-free timolol and preservative-free dorzolamide-timolol combination to control IOP. Much of the resultant damage after chemical injuries is often due to the inflammatory reaction, so suppressing it with corticosteroids is very important. Topical corticosteroids do run the risk of corneal stromal lysis after the first week, so once again systemic agents may be considered during this time frame [39]. If glaucoma surgery becomes necessary, trabeculectomy is usually not a good option due to inflamed and scarred conjunctiva. Glaucoma drainage implants and cyclodestruction are likely more viable options. Minimally invasive glaucoma surgical options are considerations, but these require visibility of the angle structures through a clear cornea. Some surgeons have described endoscopic approaches to implanting trabecular bypass implants and supraciliary implants as well performing ab interno trabeculotomy. These can be considered if they are within the surgeon's skill set.

Medical Management of Traumatic Glaucoma

The mainstay of medical therapy for elevated IOP in the setting of ocular trauma largely revolves around aqueous suppression. Topical aqueous suppressants generally start to take effect 1 hour after administration but reach their peak effect at 2 hours.

The beta adrenergic antagonists include timolol, metipranolol, levobunolol, carteolol, and betaxolol. Of these, all are non-selective beta blockers with the exception of betaxolol. The non-selective drugs should be avoided in those with a history of asthma and chronic obstructive pulmonary disease. Betaxolol is a beta-one selective drug and may be safer for patients with pulmonary problems but at the cost of being less efficacious than its non-selective brethren. Beta blockers should also be avoided in those with more than first-degree heart block and those with congestive heart failure.

Brimonidine is an alpha-two selective agent that also works primarily as an aqueous suppressant. It should not be used in infants and young children because of the risk of somnolence, hypotension, seizures, apnea, and respiratory arrest. Brimonidine is also available in a combination agent with timolol (Combigan) that may be helpful to ease the medication burden for those who require more than one agent to control their IOP. Apraclonidine is another alpha-two selective

agent but its incredibly high rate of follicular conjunctivitis and tachyphylaxis make it unsuited for anything but episodic use in the clinic to prevent IOP spikes after procedures.

Topical carbonic anhydrase inhibitors (CAI) such as dorzolamide and brinzolamide are also aqueous suppressants. They should be avoided in those with sickle cell disease being treated for elevated IOP secondary to hyphema as they can acidify the aqueous and promote sickling. Other options may be considered in patients with compromised corneal endothelium as CAI may contribute to corneal decompensation. Topical CAI are also especially helpful, in that they come in commercially available combinations with other aqueous suppressants such as a brinzolamide and brimonidine combination agent and a timolol and dorzolamide combination agent.

If topical agents are insufficient, systemic CAI such as acetazolamide and methazolamide may be considered. If given orally, they will show an effect at 1 hour and will reach peak effect at 2–4 hours. If given intravenously they will have an onset in 2 minutes and peak at 15 minutes. Sustained release acetazolamide in the form of Diamox sequels are helpful for chronic use as they can be taken just twice a day for a full day of coverage, but their use in an acute setting is not optimal because they do not reach peak effect until 3–6 hours after administration. Systemic CAI should be used with caution in sickle cell anemia as they can induce a sickle crisis especially if the patient is dehydrated. Acetazolamide is excreted unmetabolized in the urine and care must be taken in those with renal failure. Methazolamide by contrast is metabolized hepatically and thus is safer in renal failure patients. Care should also be taken in those with chronic obstructive pulmonary disease as the acidosis induced by systemic CAI may be harmful.

Hyperosmotics are another way to systemically control severe acute rises in IOP. They work by increasing blood osmolarity to create an osmotic gradient between blood and vitreous causing water to be drawn out of the vitreous to lower IOP. Their use in a traumatized eye is a bit controversial as the blood-aqueous barrier may be disrupted in the setting of trauma thereby diminishing its ability to establish a gradient. Oral glycerin can be given, which peaks rapidly in 30 minutes and has a duration of 5 hours. It may cause ketoacidosis in diabetics and should be used with caution. Intravenous mannitol also has a rapid onset within 10–20 minutes with a 5-hour duration and is excreted unmetabolized in the urine.

Miotics, under most circumstances should be avoided in the setting of trauma. A traumatized eye is already inflamed and a miotic may further disrupt the blood-aqueous barrier and promote the formation of posterior synechiae. Prostaglandins similarly have little utility in the acute setting, as their pro-inflammatory nature is not suitable in that scenario. Their utility will come once the eye is quiet to treat more chronic forms of glaucoma such as angle recession.

Surgical Management of Traumatic Glaucoma

When medical management has failed to control IOP and the optic nerve is deemed at risk, surgical intervention is required. In recent years, the number of potential surgical options has expanded dramatically. The timing of surgery and the choice of procedure depend on many factors, including the timing and the nature of the injury, the degree of IOP elevation, concomitant ocular and systemic comorbidities, conjunctival health, corneal clarity, the presence or absence of inflammation, the presence or absence of anterior synechiae, and any preexisting history of glaucoma.

Lasers

Lasers can be used in the management of glaucomas associated with trauma to decrease IOP by either improving aqueous outflow or decreasing aqueous production [40–46].

Laser Iridectomy

Laser iridectomy is indicated if pupillary block develops producing angle closure. It creates a

new pathway to allow sequestered aqueous to move from the posterior chamber to the anterior chamber bypassing the pupillary block, reversing iris bombe, and restoring outflow through the trabecular meshwork. Pupillary block in the setting of trauma can occur as a result of inflammation producing posterior synechiae or by virtue of forward movement of the lens due to zonular compromise.

Laser Trabeculoplasty

Laser trabeculoplasty (LTP) has limited utility in the setting of trauma. Compromised trabecular function in traumatic forms of glaucoma tends not to respond well to LTP. The success rate for laser trabeculoplasty in the setting of angle recession glaucoma was only 23% according to one report [30].

Cyclophotocoagulation

Continuous wave diode laser transscleral cyclophotocoagulation (TSCPC) reduces aqueous humor production and is useful when traditional glaucoma filtration surgery cannot be performed because of severe conjunctival scarring or when corneal clouding precludes the view to perform ab interno procedures. Classically, because of the risk for severe complications like persistent hypotony and phthisis, TSCPC has often been reserved for cases with very poor visual prognosis.

Micropulse laser treatment (MicroPulse P3 Glaucoma Device and Cyclo G6 Glaucoma Laser System; Iridex, Mountain View, CA) uses less total laser energy than traditional continuous-wave TSCPC. It breaks up the laser energy application into very short bursts to limit thermal damage and thus induces minimal inflammation and tissue destruction [46].

Endoscopic cyclophotocoagulation (ECP) has several advantages (Uram E2, Endo-Optiks, Little Silver, NJ). It uses a small endoscopic probe with a light source, an aiming beam, a diode laser, and a camera. It allows entry through a small, clear corneal or pars plana wound and gives direct visualization of the ciliary processes. This allows for direct treatment of the ciliary epithelium without the collateral damage to surrounding tissues characteristic of TSCPC and gives the ability to carefully titrate the laser energy to the minimum necessary to accomplish the task and minimize the risk for severe complications [47, 48].

Because of the improved safety profile of micropulse laser and ECP, as compared to traditional continuous wave TSCPC, many surgeons are using them now in the setting of eyes with good visual prognosis [49].

Traditional Incisional Glaucoma Filtration Surgery

Trabeculectomy

Trabeculectomy augmented with antimetabolites such as mitomycin C remains the most effective way to lower intraocular pressure surgically. However, it also has the highest complication rate of all surgical procedures and confers a cumulative lifetime risk for potentially sight-threatening complications such as bleb-related infections which is an important consideration in victims of trauma who are often young. The complications that are most feared with trabeculectomy are generally related to hypotony and its many associated sequelae such as choroidal effusions, suprachoroidal hemorrhage, and flat anterior chambers. When considering trabeculectomy, several factors need to be considered. Trabeculectomy requires healthy and intact conjunctiva. Eyes having suffered chemical injury to the conjunctiva or those who have undergone multiple prior intraocular surgeries that have violated the conjunctiva, such as pars plana vitrectomy and scleral buckling procedures, are likely poor candidates for trabeculectomy. Eyes that are actively inflamed after traumatic injuries are also at high risk for trabeculectomy failure and other options may be considered. But if the conjunctiva is healthy, the eye is quiet, the initial injury is remote, and a low target pressure is desired, such as in late-onset angle recession glaucoma, trabeculectomy can have an excellent chance of success [24, 50–54].

Glaucoma Drainage Devices

Glaucoma drainage devices (GDD) can generally be used with greater success in the setting of trauma than trabeculectomy. They have a survival advantage in the setting of active inflammation and less than ideal conjunctival health. Trabeculectomy can only be performed superiorly as inferior placement has an unacceptably high rate of infection. However, GDD can quite easily be placed in either inferonasal or inferotemporal quadrants, providing more options to the surgeon if the superior conjunctiva is in bad condition but the inferior is relatively healthy. If the conjunctiva is insufficient at the surgical site, conjunctival autografts or amniotic membrane grafts can be used. The tubes of these implants can be placed in the anterior chamber, the ciliary sulcus in pseudophakes, or the pars plana in those who have been vitrectomized. The most commonly used implants are the Ahmed Glaucoma Valve (AGV), the Molteno, and the Baerveldt Glaucoma Implants (BGI). The non-valved devices such as the Molteno and the BGI have the advantage of typically producing lower IOP than the AGV with lower rates of failure and of hypertensive phase due to their larger plate sizes. However, these non-valved devices have to be tied off with a ligature to prevent their immediate functioning to allow for a fibrous capsule to form around the plate of the implant to provide some resistance to aqueous outflow and prevent overfiltration and hypotony. This process takes a minimum of 3–4 weeks, so if the patient's IOP is extraordinarily high, the necessary wait may be deemed unacceptable. By contrast, the AGV has the advantage of having a valve mechanism to prevent early hypotony giving it the ability to reliably lower IOP immediately without the need for a temporary ligature. This advantage, however, comes at the cost of higher rates of hypertensive phase, higher final IOP, and higher rates of failure due to inadequately controlled IOP. The Molteno and the BGI, by contrast, have higher rates of failure due to hypotony-related complications than the AGV. The potential complications are similar to trabeculectomy, but they tend to occur less frequently. Complications unique to drainage implants include diplopia, corneal decompensation, and tube or plate erosions [31, 33, 40, 55–58].

Minimally Invasive Glaucoma Surgery

Minimally invasive glaucoma surgery or microinvasive glaucoma surgery (MIGS) is a new class of procedures that are gaining in popularity. The basic tenets of MIGS are that they are performed ab interno through small, clear corneal incisions under direct gonioscopic visualization without violating the conjunctiva. They are therefore most useful and applicable in cases where the angle is open and without peripheral anterior synechiae and the cornea is clear. These procedures have a more modest efficacy in lowering IOP when compared to trabeculectomy and glaucoma drainage implants, but they offer a greatly diminished risk for complications especially of the kind related to hypotony, infections, and erosions.

MIGS Involving Trabecular Outflow

There have been reports of the successful use of MIGS in lowering IOP in traumatic glaucoma with iStent trabecular micro-bypass stent (Glaukos, San Clemente, CA), ab interno trabeculectomy with Trabectome (Neomedix, Tustin, CA), and gonioscopy-assisted transluminal trabeculotomy (GATT). The number of traumatic glaucoma cases in these reports are small and larger studies with longer follow-up are needed to establish the efficacy of these procedures. These MIGS techniques work by bypassing the trabecular meshwork either with a bypass implant (iStent) or by ablating the trabecular meshwork (Trabectome) or tearing open the trabecular meshwork circumferentially with the GATT procedure [59–61]. Some other MIGS alternatives to accomplish this trabecular bypass now exist, such as Kahook Dual Blade (New World Medical, Rancho Cucamonga, CA) which excises a strip of trabecular meshwork and the Trab 360 (Sight Sciences, Menlo Park, CA) which is another way to perform circumferential trabeculotomy, but their effectiveness in traumatic glaucoma have not been reported.

MIGS Involving Suprachoroidal and Subconjunctival Pathways

Some newer MIGS procedures that have recently received FDA approval are the CyPass Micro-Stent (Alcon, Fort Worth, TX), an implant that shunts aqueous into the suprachoroidal space, and the Xen 45 gel stent (Allergan, Dublin, Ireland), which shunts into the subconjunctival space. Neither has been proven effective in the setting of traumatic glaucoma. CyPass shares many similarities to the other trabecular bypass MIGS procedures, in that aqueous outflow is contained within the eye and does not depend on conjunctival health. The Xen implant requires healthy conjunctiva and the application of anti-metabolites such as MMC and thus shares some of the same limitations as trabeculectomy.

References

1. Canavan YM, Archer DB. Anterior segment consequences of blunt ocular injury. Br J Ophthalmol. 1982;66:549–55.
2. Girkin CA, McGwin G, Long C, Morris R, Kuhn F. Glaucoma after ocular contusion: a cohort study of the United States Eye Injury Registry. J Glaucoma. 2005;14(6):470–3.
3. Girkin CA, McGwin G, Morris R, Kuhn F. Glaucoma following penetrating ocular trauma: a cohort study of the United States Eye Injury Registry. Am J Ophthalmol. 2005;139(1):100–5.
4. Bazzaz S, Katz JL, Myers JS. Post-traumatic glaucoma. In: Shaarawy T, editor. Glaucoma: medical diagnosis and therapy. London: Elsevier; 2009. p. 431–9.
5. Coles WH. Traumatic hyphema: an analysis of 235 cases. South Med J. 1968;61:813–6.
6. Crouch ER Jr, Crouch ER. Management of traumatic hyphema: therapeutic options. J Pediatr Ophthalmol Strabismus. 1999;36:238–50.
7. Volpe NJ, Larrison WI, Hersh PS, et al. Secondary hemorrhage in traumatic hyphema. Am J Ophthalmol. 1991;112:507–13.
8. Howard GM, Hutchinson BT, Frederick AR. Hyphema resulting from blunt trauma. Gonioscopic, tonographic, and ophthalmoscopic observations following resolution of the hemorrhage. Trans Am Acad Ophthalmol Otolaryngol. 1965;69:294–306.
9. Edwards WC, Layden WF. Traumatic hyphema. A report of 184 consecutive cases. Am J Ophthalmol. 1973;75:110–6.
10. Fong LP. Secondary hemorrhage in traumatic hyphema. Predictive factors for selective prophylaxis. Ophthalmology. 1994;101:1583–8.
11. Ganley JP, Geiger JM, Clement JR, et al. Aspirin and recurrent hyphema after blunt ocular trauma. Am J Ophthalmol. 1983;96:797–801.
12. Spoor TC, Kwito GM, O'Grady JM, et al. Traumatic hyphema in an urban population. Am J Ophthalmol. 1990;109:23–7.
13. Crouch ER, Frenkel M. Aminocaproic acid in the treatment of hyphema. Am J Ophthalmol. 1976;81:355–60.
14. Deans R, Noeel LP, Clarke WN. Oral administration of tranexamic acid in the management of traumatic hyphema in children. Can J Ophthalmol. 1992;27:181–3.
15. Rahmani B, Jahadi H. Comparison of tranexamic acid and prednisolone in the treatment of traumatic hyphema. Ophthalmology. 1999;106:375–9.
16. Nasrullah A, Kerr NC. Sickle cell as a risk factor for secondary hemorrhage in children with traumatic hyphema. Am J Ophthalmol. 1997;123:783–90.
17. Goldberg MF. Sickled erythrocytes, hyphema, and secondary glaucoma. The effect of vitamin C on erythrocyte sickling in aqueous humor. Ophthalmic Surg. 1979;10:70–7.
18. Weiss JS, Parrish RK, Anderson DR. Surgical therapy of traumatic hyphema. Ophthalmic Surg. 1983;14:343–5.
19. Walton W, Von Hagen S, Grigorian R, Zarbin M. Management of traumatic hyphema. Surv Ophthalmol. 2002;47(4):297–334.
20. Belcher CD, Brown SV, Simmons RJ. Anterior chamber washout for tramatic hyphema. Ophthalmic Surg. 1985;16(8):475–9.
21. Mooney D. Angle recession and secondary glaucoma. Br J Ophthalmol. 1973;57:608–12.
22. Spaeth GL. Traumatic hyphema, angle recession, dexamethasone hypertension, and glaucoma. Arch Ophthalmol. 1967;78:714–21.
23. Blanton FM. Anterior chamber angle recession and secondary glaucoma: a study of the after effects of traumatic hyphemas. Arch Ophthalmol. 1964;72:39–43.
24. Tumbocon JA, Latina M. Anglerecession glaucoma. Int Ophthalmol Clin. 2002;42(3):69–78.
25. Kaufman JH, Tolpin DW. Glaucoma after traumatic angle recession: a ten-year prospective study. Am J Ophthalmol. 1974;79:648–54.
26. Wolff SM, Zimmerman LE. Chronic secondary glaucoma: associated with retrodisplacement of iris root and deepening of the anterior chamber angle secondary to contusion. Am J Ophthalmol. 1962;54:547–63.
27. Herschler J. Trabecular damage due to blunt anterior segment injury and its relationship to traumatic glaucoma. Trans Am Acad Ophthalmol Otolaryngol. 1977;83:239–48.
28. Salmon JF, Mermoud A, Ivey A. The detection of post-traumatic angle recession by gonioscopy in a population-based glaucoma survey. Ophthalmology. 1994;101:1844–50.

29. Tesluk GC, Spaeth GL. The occurrence of primary open-angle glaucoma in the fellow eye of patients with unilateral angle-cleavage glaucoma. Ophthalmology. 1985;92:904–11.
30. Scharf B, Chi T, Grayson D, et al. Argon laser trabeculoplasty for angle recession glaucoma. Invest Ophthalmol Vis Sci. 1992;33(Suppl):1159.
31. Mermoud A, Salmon JF, Barron A, et al. Surgical management of post-traumatic angle recession glaucoma. Ophthalmology. 1993;100:634–42.
32. Mermoud A, Salmon JF, Straker C, et al. Post-traumatic angle recession glaucoma: a risk factor for bleb failure after trabeculectomy. Br J Ophthalmol. 1993;77:631–4.
33. Yadgarov A, Liu D, Crane E, Khouri A. Surgical outcomes of Ahmed or Baerveldt tube shunt implantation for medically uncontrolled traumatic glaucoma. J Curr Glaucoma Pract. 2017;11(1):16–21.
34. Cioffi GA, Durcan FJ, Girkin CA, Gupta N, Piltz-Seymour JR, Samuelson TW, Tanna AP, Barton K, O'Connell SS. Glaucoma. In: Skuta GL, editor. Basic and clinical science course. San Francisco: American Academy of Ophthalmology; 2012. p. 1–212.
35. Dotan S, Oliver M. Shallow anterior chamber and uveal effusion after nonperforating trauma to the eye. Am J Ophthalmol. 1982;94:782–4.
36. Samples JR, Van Buskirk EM. Open-angle glaucoma associated with vitreous humor filling the anterior chamber. Am J Ophthalmol. 1986;102:759–61.
37. Matsuo T, Muraoka N, Shiraga F, et al. Schwarz-Matsuo syndrome in retinal detachment with tears of the nonigmented epithelium of the ciliary body. Acta Ophthalmol Scand. 1998;76:481–5.
38. Paterson CA, Pfister RR. Intraocular pressure changes after alkali burns. Arch Ophthalmol. 1974;91:211–8.
39. Donshik PC, Berman MB, Dohlman CH, et al. Effect of topical corticosteroids on ulceration in alkali-burned corneas. Arch Ophthalmol. 1978;96:2117–20.
40. Shields SR, Chen P. Sequential or simultaneous cyclophotocoagulation and glaucoma drainage implant for refractory glaucoma. J Glaucoma. 2002;11(3):203–8.
41. Lieberman MF, Hoskins HD Jr, Hetherington J Jr. Laser trabeculoplasty and the glaucomas. Ophthalmology. 1983;90(7):790–5.
42. Robin AL, Pollack IP. Argon laser trabeculoplasty in secondary forms of open-angle glaucoma. Arch Ophthalmol. 1983;101(3):382–4.
43. Spaeth GL, Fellman RL, Starita RJ, Poryzees EM. Argon laser trabeculoplasty in the treatment of secondary glaucoma. Trans Am Ophthalmol Soc. 1983;81:325–32.
44. Schlote T, Grüb M, Kynigopoulos M. Long-term results after transscleral diode laser cyclophotocoagulation in refractory posttraumatic glaucoma and glaucoma in aphakia. Graefes Arch Clin Exp Ophthalmol. 2008;246(3):405–10.
45. Bloom PA, Clement CI, King A, et al. A comparison between tube surgery, Nd:YAG laser and diode laser cyclophotocoagulation in the management of refractory glaucoma. Biomed Res Int. 2013;2013:371951.
46. Aquino MC, Barton K, Tan AM, et al. Micropulse versus continuous wave transscleral diode cyclophotocoagulation in refractory glaucoma: a randomized exploratory study. Clin Exp Ophthalmol. 2015;43(1):40–6.
47. Pantcheva MC, Kahook MK, Schuman JS, Noecker RJ. Comparison of acute structural and histopathological changes in human autopsy eyes after endoscopic cyclophotocoagulation and transscleral cyclophotocoagulation. Br J Ophthalmol. 2007;91:248–52.
48. Lin SC, Chen MJ, Lin MS, Howes E, Stamper RL. Vascular effects of ciliary tissue from endoscopic versus trans-scleral cyclophotocoagulation. Br J Opthalmol. 2006;90:496–500.
49. Yang Y, Zhong J, Dun Z, Liu XA, Yu M. Comparison of efficacy between endoscopic cyclophotocoagulation and alternatie surgeries in refractory glaucoma: a meta-analysis. Medicine (Baltimore). 2015;94(39):e1651.
50. Joseph JP, Miller MH, Hitchings RA. Wound healing as a barrier to successful filtration surgery. Eye. 1988;2:5113–23.
51. AGIS Investigators. The Advanced Glaucoma Intervention Study (AGIS): 11. Risk factors for failure of trabeculectomy and argon laser trabeculoplasty. Am J Ophthalmol. 2002;134(4):481–98.
52. Mermoud A, Salmon JF, Straker C, et al. Post-traumatic angle recession glaucoma risk factor for bleb failure after trabeculectomy. Br J Ophthalmol. 1993;77:651–4.
53. Manners T, Salmon JF, Barron A, Willies C, Murray DN. Trabeculectomy with mitomycin C in the treatment of post-traumatic angle recession glaucoma. Br J Ophthalmol. 2001;85:159–63.
54. Turalba AV, Shah AS, Andreoli MT, Andreoli CM, Rhee DJ. Predictors and outcomes of ocular hypertension after open-globe injury. J Glaucoma. 2014;23(1):5–10.
55. Mermoud A, Salmon JF, Straker C, et al. Predictors and outcomes of ocular hypertension after open-globe injury. J Glaucoma. 2014;23(1):5–10.
56. Nguyen QH. Primary surgical management refractory glaucoma: tubes as initial surgery. Curr Opin Ophthalmol. 2009;20(2):122–5.
57. Fuller JR, Bevin TH, Molteno AC. Long-term follow-up of traumatic glaucoma treated with Molteno implants. Ophthalmology. 2001;108(10):1796–800.
58. Budenz DL, Barton K, Gedde SJ, Feuer WJ, Schiffman J, Costa VP, Godfrey DG, Buys YM. Five-year treatment outcomes in the Ahmed Baerveldt comparison study. Ophthalmology. 2015;122(2):308–16.
59. Jordan JF, Wecker T, van Oterendorp C, et al. Trabectome surgery for primary and secondary open angle glaucomas. Graefes Arch Clin Exp Ophthalmol. 2013;251(12):2753–60.
60. Buchacra O, Duch S, Milla E, Stirbu O. One-year analysis of the iStent trabecular microbypass in secondary glaucoma. Clin Ophthalmol. 2011;5:321–6.
61. Grover D, Godfrey D, Smith A, et al. Gonioscopy-assisted transluminal trabeculotomy, ab interno trabeculotomy technique report and preliminary results. Ophthalmology. 2014;121(4):855–61.

Periocular and Orbital Trauma

9

Raymond I. Cho and Sheri L. DeMartelaere

Introduction

Injuries to the periocular structures and orbit are an extremely common component of ocular trauma. Historical reports indicate a 10–20% incidence of orbital fractures among all combat casualties from Vietnam, Iraq, and Afghanistan [1, 2]. During Operation Iraqi Freedom, eyelid lacerations, orbital fractures, and retrobulbar hemorrhage were seen in 21–36% of patients with combat ocular trauma [3, 4]. As can be said of all trauma, no two cases are the same, and management decisions and surgical techniques must be tailored to each individual patient. The goal of this chapter is not to prescribe precise decision-making algorithms and treatment methods, but rather to provide general guidelines, insights, and caveats to consider when managing these often complex cases. In all cases, however, the overarching principles of trauma surgery still apply.

With the notable exception of orbital compartment syndrome, most oculoplastic trauma can be managed on a non-emergent basis. Ideally, lacerations of the periocular soft tissue should be repaired within 24 hours to reduce the risk of infection, allow for anatomic approximation, and facilitate wound healing. Most orbital fractures can be safely observed for days or even weeks in the absence of extraocular muscle entrapment. Burn injuries can often be managed conservatively for weeks to months before surgical intervention is undertaken. As with most traumatic injuries, consideration should be given to address potential foreign bodies and contamination through preoperative and/or intraoperative antibiotics, tetanus prophylaxis, wound exploration, and irrigation with sterile saline solution.

Eyelid Trauma

Partial-Thickness Lacerations

Eyelid lacerations that involve only the skin and orbicularis oculi muscle (the anterior lamella) and do not violate the tarsus or orbital septum are considered partial thickness. Simple linear non-stellate lacerations can be closed with superficial interrupted or running sutures. Monofilament sutures are generally preferred superficially and can be absorbable or non-absorbable based on surgeon preference and the likelihood that the patient will follow up for suture removal. In uncomplicated lacerations with good vascular supply and low patient risk factors, 5–7 days is generally sufficient time for sutures to be left in place. When absorb-

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able sutures are preferred, plain gut or fast-absorbing plain gut are typically used; 6-0 is the most common suture size used in eyelid skin. For stellate or curvilinear lacerations, it is critical to ensure that there is no loss of tissue and that the flaps of skin are re-approximated as anatomically as possible. It is advisable to minimize debridement of eyelid tissue, even if it appears to be devitalized, since the excellent vascular supply of the periocular region often allows such tissue to survive. If significant wound tension is encountered due to tissue edema or other factors, buried sutures can be placed in the deep dermis or orbicularis muscle to alleviate the tension on the skin. Absorbable sutures are generally utilized for this purpose, with the size and material tailored to the situation.

Full-Thickness Lacerations

Lacerations that violate the tarsus or orbital septum are considered full thickness. Prolapse of orbital fat through the wound is an indication of septal violation. The involvement of septum itself is not a concern, but it does raise the possibility of injury to the globe and other vital orbital structures. The structure most likely to be injured by virtue of its proximity to the septum is the aponeurosis of the levator palpebrae superioris muscle. The wound should be carefully explored for evidence of damage to the aponeurosis, and if a laceration is clearly found, it can be repaired primarily. However, if the levator aponeurosis cannot be identified beyond a shadow of a doubt, it should be left alone or referred to an oculoplastic surgeon. The pitfall of repairing tissue deep to the anterior lamella is that septum can be easily mistaken for levator and, if sutured, can lead to postoperative lid retraction and lagophthalmos. *Under no circumstances should orbital septum ever be repaired with sutures or other means.*

Lacerations that involve the lid margin and tarsus require meticulous repair to restore the proper function and contour of the eyelid. The tarsal wound edges should be carefully explored for evidence of tissue loss or maceration. If the tissue appears unhealthy or the tarsal segments do not perfectly align with one another, the edges should be freshened with a scalpel blade or sharp scissors to create proper tarsal alignment and contour of the margin. The tarsus should then be repaired with two or three braided absorbable or non-absorbable sutures at the lid margin in a simple interrupted or vertical mattress fashion (Fig. 9.1a). Proper alignment of the mucocutaneous junction, gray line, and lash line is critical. The rest of the tarsus should be repaired with interrupted braided absorbable sutures passed partial thickness through the tarsus so as to avoid abrading the ocular surface (Fig. 9.1b). The orbicularis and skin can then be closed as described for partial-thickness lacerations. Lid margin sutures are typically left in place for at least 10–14 days.

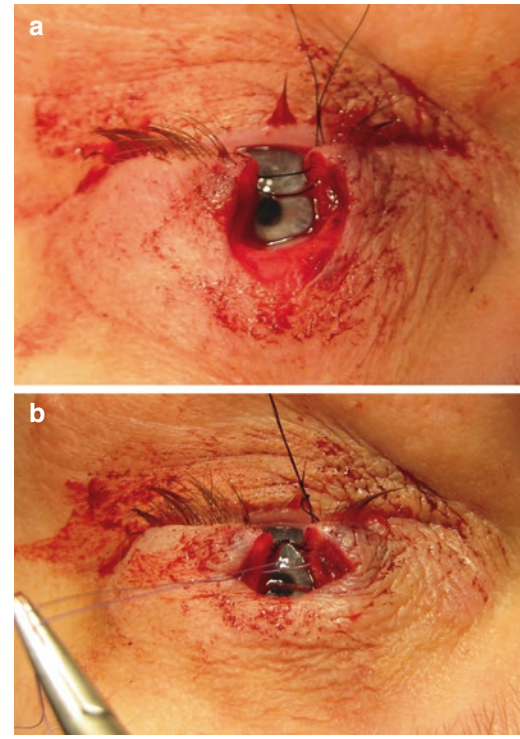

Fig. 9.1 Full-thickness upper lid laceration repair following pentagonal wedge resection. (**a**) The initial margin suture is placed through the mucocutaneous junction with a 6-0 silk suture in a vertical mattress fashion. (**b**) The remainder of the tarsus is repaired with partial-thickness 6-0 Vicryl sutures. (Reprinted with permission from Savitsky and Eastridge [28])

Periocular Tissue Loss

Traumatic loss of periocular tissue is uncommon. It is more common for complex full-thickness lid

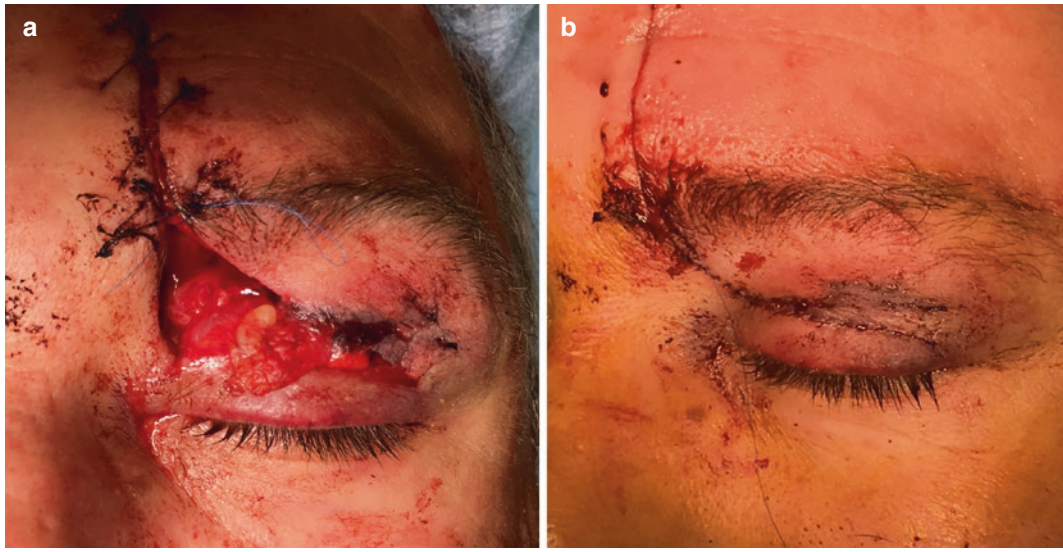

Fig. 9.2 Degloving injury of the left forehead and upper eyelid. (**a**) Preoperative appearance suggests significant soft tissue loss over the medial upper lid. (**b**) Careful wound exploration and approximation reveals preservation of all upper lid tissue

lacerations to create the appearance of significant tissue loss, due to the tendency of the wound edges to retract and curl underneath themselves. Careful wound exploration in these cases can reveal the presence of tissue previously thought to be missing (Fig. 9.2). In rare cases of full-thickness eyelid avulsion in which the lid segment is recovered, it is often possible for the tissue to be successfully replanted [5]. When true tissue loss occurs, there are numerous options for reconstruction, a full discussion of which is beyond the scope of this text. As a general rule, it is advisable in the setting of acute trauma to perform the minimum necessary surgery to salvage remaining tissue, prevent infection, and protect the globe until delayed reconstruction can be performed in a more controlled fashion on a more stable wound bed. The temptation to immediately perform such complex procedures as Hughes flaps, Mustarde flaps, or paramedian forehead flaps should be avoided.

Burns

The goal of periorbital burn management is to preserve vision by preventing eyelid retraction resulting in ectropion, chronic exposure of the ocular surface, desiccation, ulceration, and permanent corneal scarring. The majority of periorbital burns are managed conservatively with standard ocular surface lubrication used in the setting of any sedated intubated patient [6]. However, patients that have sustained deep partial second-degree burns and deeper may at some time require either early or late surgical intervention. All patients with periorbital burns should receive ophthalmology consultation.

Although the literature is not uniformly definitive, third- and fourth-degree burns may benefit from acute debridement with full-thickness skin grafting. In patient's where adequate tissue for skin grafting is not available or the patient is medically unstable, temporizing measures such as more aggressive lubrication, suture tarsorrhaphy, and moisture chamber goggles may be utilized. Allogeneic skin substitutes may also be a viable alternative. In those patients with rapidly evolving eyelid retraction and ectropion, other ocular surface temporizing measures such as PROKERA® and PROSE® may extend the timeline for observation before surgery is required [7]. It has been documented [6, 8] that those patients that require surgery often require more than one surgical intervention.

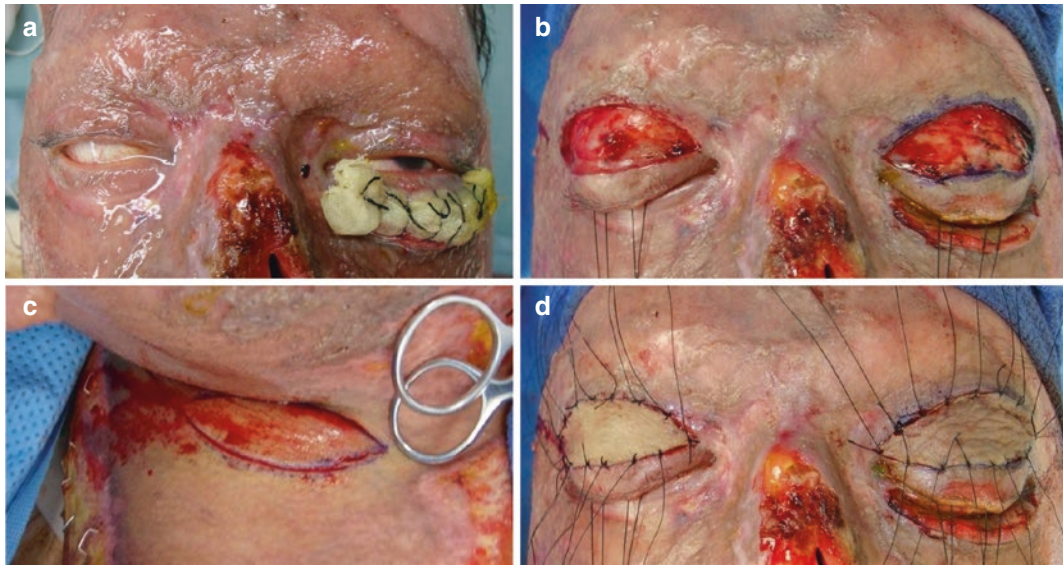

Fig. 9.3 Periocular burn injury. (**a**) Preoperative photograph showing bilateral upper lid retraction and lagophthalmos (bolster over the left lower lid from previous skin graft). (**b**) Upper lid scar release at the level of the upper lid crease. (**c**) Full-thickness skin graft harvested from the right supraclavicular region. (**d**) Skin grafts over both upper lids prior to bolster placement

A delay in surgery might enable sufficient time for a keratinocyte autograft or the ability to reserve the best possible donor site for the definitive procedure.

Surgical reconstruction of the periocular burn patient can involve a number of techniques, the most common of which is full-thickness skin grafting (Fig. 9.3). Split-thickness skin grafts are not optimal for periocular reconstruction due to their propensity for contracture but may be warranted if the donor skin is particularly thick. The ideal skin graft is thin, non-hair bearing, and matches the skin color of the host site. Common donor sites include the contralateral upper lid, preauricular, retroauricular, supraclavicular, volar upper arm, thigh, and abdomen. In severe burn cases with large body surface area involvement, however, the choices may be slim. Other options for periocular reconstruction of the anterior lamella include local advancement, transpositional, or interpolated flaps, depending on the degree of involvement of the surrounding tissues. Posterior lamellar reconstruction is not often required, but options can include autogenous grafts such as conjunctiva, tarsus/conjunctiva, buccal mucous membrane, hard-palate mucosa, dermis-fat grafts, auricular cartilage, and nasal mucosa; local flaps such as temporoparietal fascia, temporalis muscle, pericranium, periosteal flaps; and allografts or xenografts such as human acellular dermis and fascia lata. Alloplastic implants should be avoided if possible, due to the high likelihood of vascular compromise in the surrounding soft tissue and the risk of infection.

For patients undergoing aggressive fluid resuscitation, the possibility of the development of orbital compartment syndrome (see orbital trauma section) necessitates close monitoring in the early post-burn period. These patients can often be difficult to assess as they tend to be intubated and sedated rendering one unable to assess vision, pain, and motility. Furthermore, the pupils are typically pinpoint masking an early APD. If in doubt, lateral canthotomy and cantholysis should be performed at the earliest opportunity.

Lacrimal Trauma

Canalicular Lacerations

Involvement of the canalicular system is common with lacerations of the medial lower and upper lids (Fig. 9.4). The mechanism of injury can be sharp or blunt and is often seen in the setting of medial canthal tendon avulsion. Dog bites are a particularly common cause [9]. The timing of repair is not urgent, but it should ideally be performed within 24–48 hours. Preservation of lacrimal flow requires stenting of the involved canaliculus in conjunction with surgical repair [10]. The most common material used for stenting is silicone, and the stent can be placed through the involved canaliculus only (monocanalicular), through both upper and lower canaliculi (bicanalicular), or through the entire nasolacrimal drainage system. While the stent need only extend as far as the lacerated portion of canaliculus, bicanalicular and nasolacrimal stents tend to be more secure postoperatively. Once the stent is in place, the cut ends of the canaliculus are approximated with fine absorbable braided sutures, and the remainder of the lid laceration is repaired as appropriate. If the medial canthal tendon is avulsed, it should be resuspended prior to repairing the canaliculus to restore proper lid position and take undue tension off the repair. Canalicular stents are typically removed at 3–6 months postoperatively.

Nasolacrimal Duct Injury

Injury to the lacrimal sac and nasolacrimal duct can occur with naso-orbital-ethmoid fractures or other midface fractures; however, these injuries are not usually directly addressed beyond repair of the associated fracture. Some authors advocate stenting of the nasolacrimal duct in such cases [11]. However, it is more typical to observe such cases for the development of epiphora and perform delayed dacryocystorhinostomy if necessary.

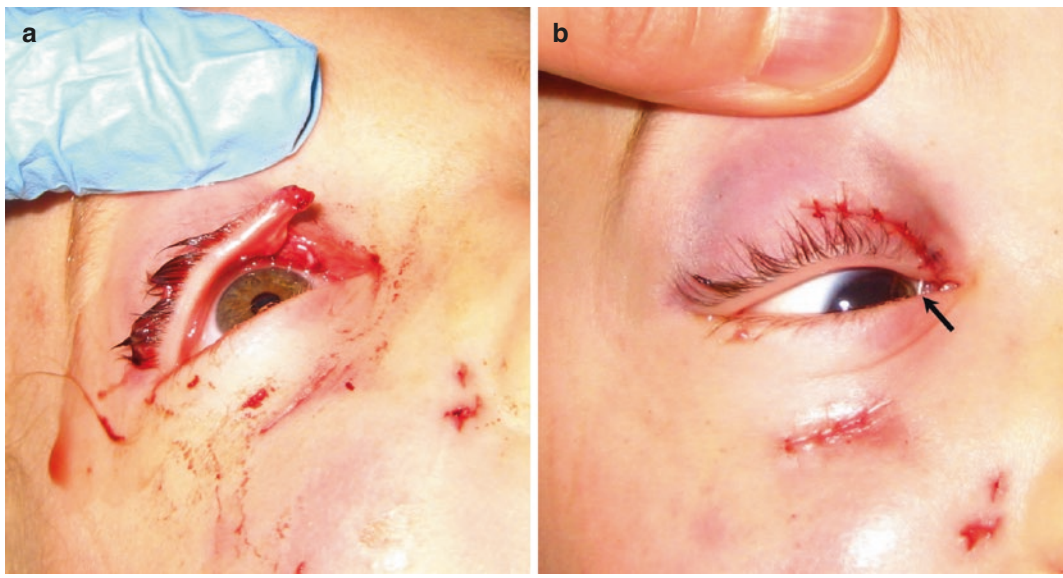

Fig. 9.4 Dog bite injury. (**a**) Preoperative photograph showing full-thickness medial upper lid laceration with avulsion of the medial canthal tendon and canalicular laceration. (**b**) Postoperative photograph following repair of medial canthal tendon avulsion and plastic repair of the upper canaliculus with a bicanalicular silicone stent (arrow)

Orbital Trauma

Orbital Compartment Syndrome

Orbital compartment syndrome occurs when the intraorbital pressure exceeds the perfusion pressure of the ophthalmic artery. Traumatic retrobulbar hemorrhage is the most common cause, but other causes include third spacing of fluid following massive fluid resuscitation in burn patients, orbital emphysema (such as that caused by forceful nose blowing in the presence of an orbital blowout fracture), and crush injury. Signs and symptoms of orbital compartment syndrome include acute vision loss, eye pain, headache, proptosis, resistance to retropulsion, increased intraocular pressure, afferent pupillary defect, ophthalmoplegia, and hemorrhagic chemosis. While imaging studies can help to identify the cause and confirm the presence of retrobulbar hemorrhage (Fig. 9.5), the diagnosis of orbital compartment syndrome is clinical, and treatment should not be delayed to obtain a computerized tomography (CT) scan. When clinical suspicion exists, lateral canthotomy and inferior cantholysis should be performed immediately [12]. Timing is critical, as experimental studies suggest that permanent vision loss occurs with orbital ischemia lasting greater than 100 minutes [13].

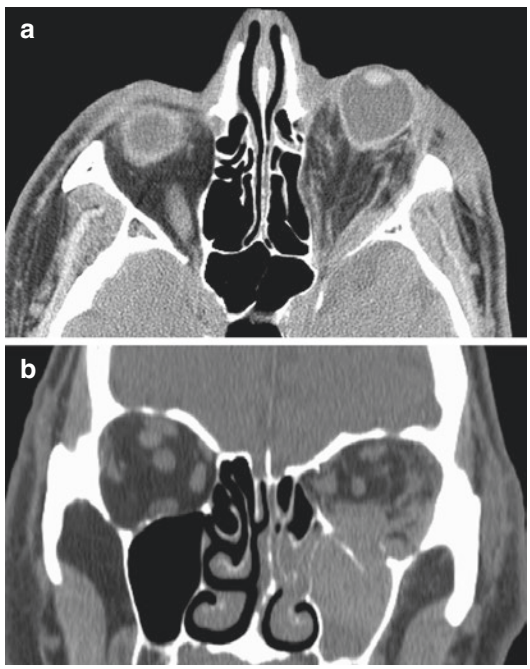

Fig. 9.5 (**a**) axial and (**b**) coronal CT scan images of patient with retrobulbar hemorrhage and orbital compartment syndrome due to blunt trauma. Note the tenting deformity of the posterior globe in the axial view

The goal of lateral canthotomy and inferior cantholysis is to convert the orbit from a closed to an open compartment. Significant drainage of blood from the orbit is not necessary for the procedure to be effective, nor is it expected. However, if the decompressive effect from the inferior cantholysis is insufficient, a superior cantholysis can be performed by releasing the upper lid in the same manner as the lower lid. The efficacy of the treatment can be assessed soon after the procedure through repeated intraocular pressure measurements and visual acuity assessment.

If orbital ischemia persists despite complete inferior and superior cantholysis, additional maneuvers may be attempted. Blunt scissors can be placed through the canthotomy incision and be used to spread the orbital fat in the inferotemporal quadrant, releasing any pockets of loculated blood within the orbit [14]. If this maneuver fails, the patient may be taken to the operating room for formal orbitotomy and decompression by whatever means necessary. Medical therapy with intraocular pressure-lowering agents such as osmotics or aqueous suppressants may also be considered.

The importance of canthotomy and cantholysis in the setting of orbital compartment syndrome cannot be overstated. There is no other procedure in all of ophthalmology that can more quickly and dramatically impact a patient's final visual outcome. Performed promptly enough, it can literally mean the difference between no-light-perception and 20/20 vision.

Orbital Foreign Bodies

Penetrating orbital injury from projectiles or stab injuries can result in retained orbital foreign bodies. Patients typically present with varying degrees of periocular edema/ecchymosis,

proptosis, impaired ocular motility, decreased vision, and pain. The size, location, and composition of the object are important in predicting its long-term tolerability within the orbit and determining the need for removal. Large objects protruding from the orbit, and those posing a risk to visual function due to their size or location should be removed. Organic foreign bodies pose a high risk of infection and inflammation, and their removal is recommended. On the other hand, many inorganic foreign bodies are inert and can be safely observed, including most metallic foreign bodies, with the exceptions of iron, copper, and lead [15]. Indications for orbital foreign body removal include vision loss, proptosis, diplopia, cellulitis, inflammation, or chronic draining fistula. Potential benefits of surgery must be weighed against the risks, which in the case of orbital surgery can be substantial due to the inherently challenging nature of operating within a tightly enclosed fat-filled space containing numerous vital structures. The use of intraoperative fluoroscopy can be considered for more rapid localization of orbital foreign bodies [16].

Orbital Floor and Medial Wall Fractures

The floor and medial wall are the thinnest and most commonly fractured walls of the orbit. These fractures can be isolated ("blowout" fractures) or associated with fractures of the facial buttresses ("blow-in" fractures). In the acute setting, the most critical factor affecting management is the presence or absence of extraocular muscle entrapment. Trap door fractures are more common in children and young adults and are typically small in size radiographically. CT scan can reveal the presence of a fracture but often fails to indicate whether or not a muscle is entrapped. Clinical signs of entrapment include diplopia, pain which increases with extraocular movement, nausea, and restricted movement both towards and away from the entrapped muscle. Some clinicians advocate forced duction testing, which can be helpful to rule out entrapment but is difficult to perform in an awake patient. If entrapment is suspected, surgical intervention should be performed as soon as practicable, ideally within 24 hours. Rarely, trap door fractures can induce the oculocardiac reflex, which manifests with bradycardia or even asystole and necessitates emergent surgery.

The majority of orbital fractures do not require repair. The most common indications for surgery are symptomatic diplopia and globe malposition (enophthalmos or globe dystopia) (Fig. 9.6) [17]. Absent true muscle entrapment, diplopia can result from either orbital edema and/or muscle contusion, which resolve over time, or herniation of orbital contents into the adjacent sinus, which may or may not resolve spontaneously. Because diplopia can resolve without treatment in many cases, some surgeons tend to observe patients for several weeks before electing to repair. Globe malposition is a function of fracture size, bony displacement, and orbital tissue herniation. Because orbital edema is so common in acute trauma, enophthalmos can take some time to become evident, leading some surgeons to again observe patients for several weeks to allow the edema to resolve and any enophthalmos to manifest itself before recommending surgery.

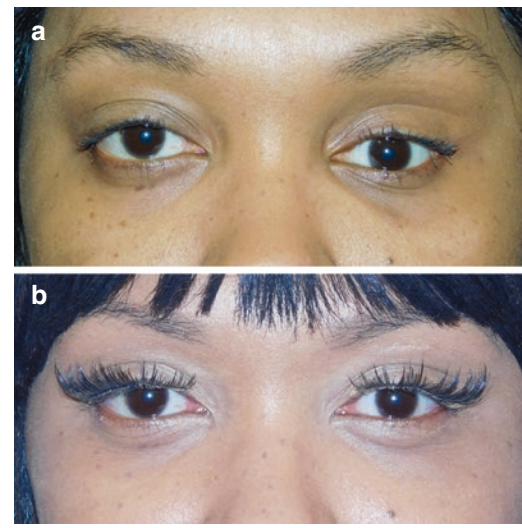

Fig. 9.6 (**a**) Preoperative photograph of patient with left-sided globe dystopia and enophthalmos following orbital floor fracture. (**b**) Postoperative photograph following orbital floor fracture repair showing restoration of normal globe position

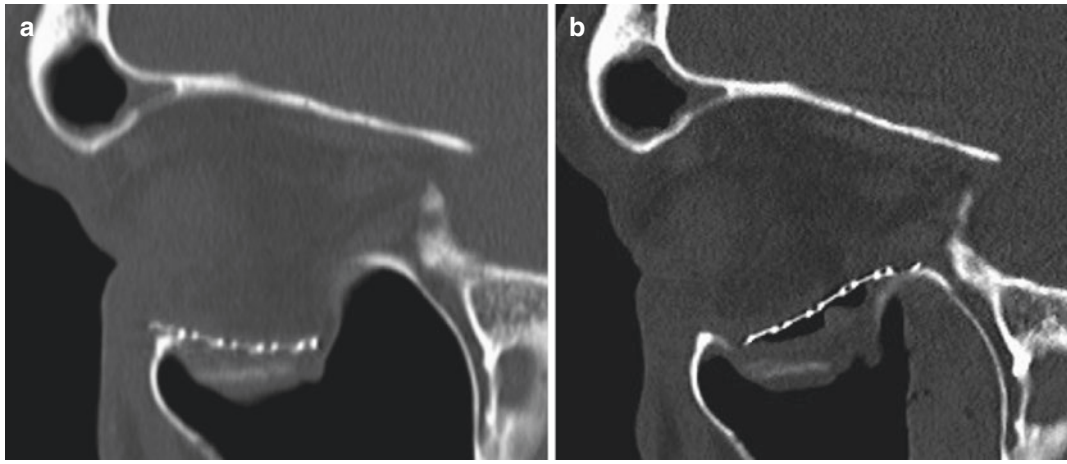

Fig. 9.7 (**a**) Sagittal CT scan image following orbital floor fracture repair with non-anatomic placement of the implant, which fails to reach the posterior ledge. (**b**) Proper implant placement on the posterior ledge following floor fracture revision. Note the gentle S-shaped curvature of the implant replicating the normal contour of the orbital floor

Orbital floor fractures are usually approached through an inferior transconjunctival or subciliary incision. Use of the orbital rim incision is highly discouraged. The transcaruncular approach is generally preferred for medial wall fractures, but the Lynch incision has also historically been used. Many implant types are available for orbital fracture repair, including but not limited to porous polyethylene, titanium, titanium embedded in porous polyethylene, nylon foil, L-poly-lactic acid, and autogenous or allogeneic bone grafts. The key to surgical success is intimate knowledge of orbital anatomy and proper surgical technique, particularly regarding implant sizing, shaping, and positioning. Implants must replicate the shape and contour of the orbital wall and rest on stable ledges of bone, especially the posterior ledge (Fig. 9.7). Improper surgical technique and/or implant positioning can result in a myriad of complications, including vision loss, diplopia, pain, numbness or paresthesias, globe malposition, eyelid malposition, and disfigurement.

Orbital Roof Fractures

The orbital roof is very strong and does not fracture easily. Unless very large and significantly displaced, most roof fractures are not repaired.

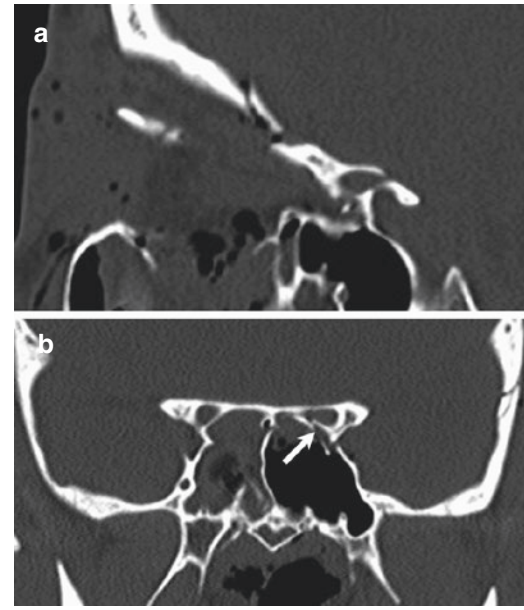

Fig. 9.8 (**a**) Sagittal CT scan image of severely comminuted fracture of the orbital roof and floor. (**b**) Coronal CT scan image of the same patient showing optic canal fracture (arrow) associated with traumatic optic neuropathy

Complications of orbital roof fractures can include proptosis, traumatic optic neuropathy (Fig. 9.8), frontal sinus fractures, cerebrospinal fluid leak, and encephalocele [18]. It has been noted that relatively small roof fractures in children can result in encephalocele [19]. When

indicated, surgical management should be conducted in conjunction with neurosurgery.

Zygomaticomaxillary Complex Fractures

Some orbital fractures are associated with fractures of the facial buttresses, one of the most common patterns being the zygomaticomaxillary complex (ZMC) or tripod/trimalar fracture. ZMC fractures classically involve the lateral orbital rim, inferior orbital rim, and zygomatic arch (Fig. 9.9). Fractures of the orbital floor and lateral orbital wall are also invariably present. In addition to the aforementioned orbital complications, other clinical signs can include facial deformity (malar flattening or widening), trismus, and lateral canthal dystopia. In contrast with the timing of isolated blowout fractures, clinically significant ZMC fractures should ideally be repaired within a week before scarring and bone healing increase the difficulty of fracture reduction.

Repair of ZMC fractures often utilizes a combination of approaches, including gingivobuccal sulcus, transconjunctival, and lateral canthotomy or lid crease incisions. The Gillies approach is also useful for depressed zygomatic arch fractures. In some cases, placement of a Carroll-Girard screw can greatly aid in mobilization and fracture reduction. Proper reduction of the zygomatic segment will typically reduce the lateral orbital wall fracture, which is an excellent landmark to indicate appropriate overall alignment of the zygomatic complex. In many cases, the orbital floor will also align sufficiently with reduction of the ZMC, unless significant comminution or orbital herniation exists preoperatively [20]. In such cases, it is often unnecessary to explore and repair the orbital floor.

Naso-Orbital-Ethmoid Fractures

Naso-orbital-ethmoid (NOE) fractures classically involve the nasal bones, maxillary frontal process, and medial orbital wall (Fig. 9.10). The

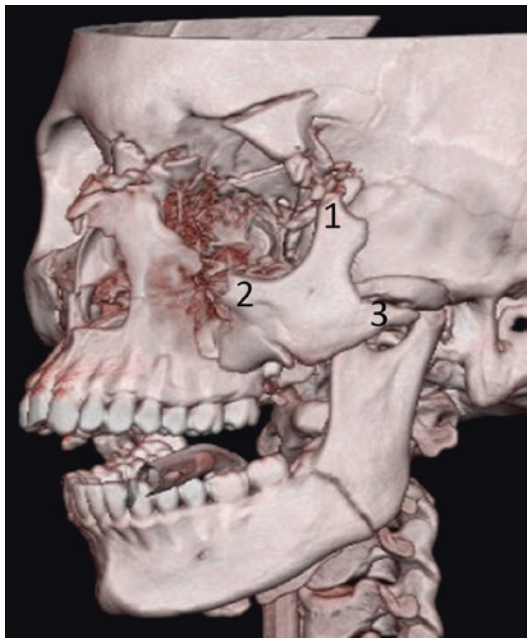

Fig. 9.9 Three-dimensional CT scan reconstruction of left zygomaticomaxillary complex fracture involving the frontozygomatic suture (1), inferomedial orbital rim (2), and zygomatic arch (3). Naso-orbital-ethmoid and mandibular fractures are also present

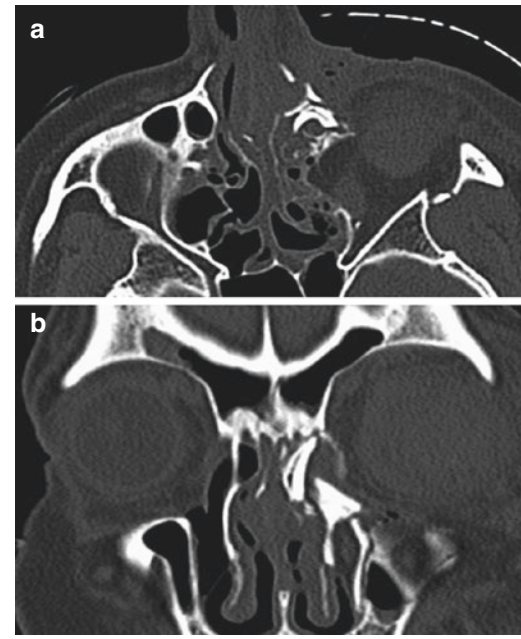

Fig. 9.10 (**a**) Axial and (**b**) coronal CT scan views of left naso-orbital-ethmoid fracture. Note the damage to the nasolacrimal duct

Markowitz classification indicates the severity of the fracture and involvement of the medial canthal tendon [21]. Complications of NOE fractures can include telecanthus, lacrimal outflow obstruction, flattening of the nasal bridge, and cerebrospinal fluid leak. Surgical repair can entail open reduction and internal fixation, transnasal wiring, and/or medial canthal tendon reinsertion. As noted previously, damage to the nasolacrimal duct often accompanies NOE fractures.

Carotid-Cavernous Fistula

The most common cause of carotid-cavernous fistula (CCF) is traumatic rupture of the intracavernous portion of the internal carotid artery. Retrograde flow of arterial blood into the superior ophthalmic vein leads to congestion of the orbit, pulsatile proptosis, ophthalmoplegia, increased intraocular pressure, episcleral venous engorgement, and conjunctival chemosis [22]. A dilated superior ophthalmic vein can be seen on orbital imaging, and angiography establishes the diagnosis (Fig. 9.11). Endovascular embolization of the fistula is typically performed by interventional radiology or neurosurgery.

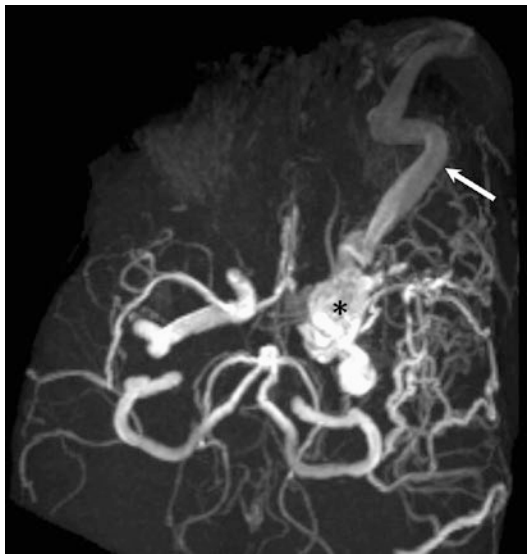

Fig. 9.11 Angiography, axial view, showing traumatic left carotid-cavernous fistula. Note the arterialization of the cavernous sinus (asterisk), dilated superior ophthalmic vein (arrow), and extensive collateral circulation

Globe Removal

Enucleation

In cases of severe globe trauma associated with blindness, pain, or risk of sympathetic ophthalmia, removal of the globe can be considered. In rare cases, globe rupture can be so severe that primary enucleation is required [23]. In the setting of trauma, many surgeons feel that enucleation is the most appropriate method of globe removal, as it theoretically minimizes the risk of sympathetic ophthalmia. Enucleation is usually performed under general anesthesia, although it can be performed under sedation with a retrobulbar block if necessary. A 360° peritomy is performed and the conjunctiva and Tenon's capsule is dissected from the scleral surface. The extraocular muscles are detached from the globe, and the optic nerve is cut posterior to the globe. After appropriate hemostasis, an orbital implant is placed within the muscle cone, and the rectus muscles are attached to the implant with braided absorbable or nonabsorbable sutures. The posterior and anterior Tenon's capsule and conjunctiva are closed over the implant with absorbable sutures, and a conformer is placed in the socket. A temporary suture tarsorrhaphy and pressure patch can be placed if desired.

The options for orbital implant materials, shapes, and sizes are numerous. Porous and non-porous materials are available, and each has its unique advantages and disadvantages. The implant should be sized to replace the majority of the globe volume while leaving enough room for an ocular prosthesis. A useful rule of thumb for spherical implants is for the diameter to be 2 mm less than the axial length of the globe [24]. In cases where placement of an alloplastic implant is inadvisable, such as the presence of gross contamination or tissue loss, a dermis-fat graft can be considered as an alternative.

Postoperatively, the patient should be instructed on conformer wear and referred to an ocularist for prosthesis fitting approximately 6 weeks later. Counseling on adjusting to life with monocular vision and protection of the remaining eye with polycarbonate lenses is also essential.

Evisceration

Removal of the intraocular contents with sparing of the scleral shell can be considered as an alternative to enucleation. As with all eviscerations, the presence of an intraocular malignancy must be ruled out preoperatively, which can be difficult in the presence of post-traumatic posterior segment changes such as retinal detachment or vitreous hemorrhage. Advantages of evisceration over enucleation include its higher tolerability under monitored anesthesia, greater ease of surgery with less tissue disruption, and improved socket motility postoperatively [25]. The issue of whether sympathetic ophthalmia is more likely to develop after evisceration compared to enucleation is controversial [26]. The procedure begins with removal of the cornea at the limbus, followed by removal of the intraocular contents with an evisceration spoon. The inside of the scleral shell is cleaned with absolute alcohol and thoroughly rinsed. Anterior and posterior sclerotomies are created with a scalpel and/or scissors to accommodate the implant, and the implant is placed within the scleral shell or in a retroscleral position within muscle cone after transecting the optic nerve [27]. For retroscleral implants, both the posterior and anterior sclerotomies are closed over the implant, while only the anterior sclerotomy is closed if the implant is placed within the scleral shell. The conjunctiva and Tenon's are then mobilized and closed over the sclera. Postoperative care and counseling is identical to the enucleation patient.

Exenteration

Removal of the globe and orbital contents is rarely indicated in the setting of trauma. Exenteration could be considered in cases of severe tissue loss or necrosis or life-threatening orbital infection. Following removal of the affected tissues, the socket can be reconstructed with flaps and/or grafts as indicated. If the eyelids and conjunctiva are preserved, it may be possible to reconstruct the socket in such a way to allow for the wear of an ocular prosthesis. If not, the socket can be filled with a vascularized free flap (closed socket) or lined with a split-thickness skin graft (open socket) to accommodate an orbital prosthesis in the future.

Conclusion

Military trauma, especially combat trauma, differs from typical civilian trauma in that it tends to be more severe, complex, and multi-system. However, with the worldwide proliferation of terrorist organizations and their targeting of civilian populations, the distinction between military and civilian trauma is becoming more blurred. High-caliber ballistic rounds, fragmentation weapons, and powerful explosives are more likely than motor vehicle accidents, pistols, and altercations to cause severe tissue damage and loss, multiple foreign bodies with gross contamination, and damage to multiple organs. For the sake of simplicity, different types of trauma are addressed individually in this chapter. However, in the combat setting, it is far more likely than not to encounter in the same patient a number of the issues discussed above, as well other types of ocular injury. Therefore, the surgeon must prioritize the treatment of each condition and consider the impact that one injury might have on another.

For example, if a patient presents with an open globe, a floor fracture, and a ZMC fracture, the open globe clearly takes priority. Once the globe is repaired, however, attempting to repair the floor fracture in the early postoperative period would place the globe repair at great risk. The ZMC, on the other hand, could safely be repaired within the first week so long as undue pressure on the globe is avoided. While most surgeons would prefer to repair both the ZMC and floor under the same anesthetic, in this case it may be more advisable to delay the floor repair until the globe is sufficiently healed. If that same patient also presented with a retrobulbar hemorrhage and a full-thickness lid laceration, one should consider the decompressive effect of the lid laceration, and either delay repair of the laceration or perform a lateral canthotomy/cantholysis before attempting primary repair.

It is clearly not possible to cover every possible situation in this text. Above all, ocular trauma surgeons must remember basic surgical principles, know the anatomy well, be flexible and creative when the situation calls for such, and not hesitate to ask for help when multidisciplinary involvement is indicated.

References

1. Hornblass A. Ocular war injuries in South Vietnam. Surg Forum. 1973;24:500–2.
2. Wade AL, Dye JL, Mohrle CR, Galarneau MR. Head, face, and neck injuries during Operation Iraqi Freedom II: results from the US Navy-Marine Corps Combat Trauma Registry. J Trauma. 2007;63:836–40.
3. Thach AB, Johnson AJ, Carroll RB, et al. Severe eye injuries in the war in Iraq, 2003-2005. Ophthalmology. 2008;155:377–82.
4. Cho RI, Kahana A, Patel B, et al. Orbital foreign body removal guided by intraoperative fluoroscopy. Ophthal Plast Reconstr Surg. 2009;25:215–8.
5. Golberg SH, Bullock JD, Connelly PJ. Eyelid avulsion: a clinical and experimental study. Ophthal Plast Reconstr Surg. 1992;4:256–61.
6. Cabalag MS, Wasiak J, Paul E, et al. Risk factors for ocular burn injuries requiring surgery. J Burn Care Res. 2017;38:71–7.
7. Czyz CN, Kalwerisky K, Stacey AW, et al. Initial treatment of ocular exposure and associated complications in severe periorbital thermal injuries. J Trauma. 2011;71:1455–9.
8. O'Connor EF, Frew Q, Din A, et al. Periorbital burns – a 6 year review of management and outcome. Burns. 2015;41:616–23.
9. Savar A, Kirszrot J, Rubin PAD. Canalicular involvement in dog bite related eyelid lacerations. Ophthal Plast Reconstr Surg. 2008;24:296–8.
10. Reifler DM. Management of canalicular lacerations. Surv Ophthalmol. 1991;36:113–32.
11. Spinelli HM, Shapiro MD, Wei LL, et al. The role of lacrimal intubation in the management of facial trauma and tumor resection. Plast Reconstr Surg. 2005;115:1871–6.
12. Yung CW, Moorthy RS, Lindley D, et al. Efficacy of lateral canthotomy and cantholysis in orbital hemorrhage. Ophthal Plast Reconstr Surg. 1994;10:137–41.
13. Hayreh SS, Kolder HE, Weingeist TA. Central retinal artery occlusion and retinal tolerance time. Ophthalmology. 1980;87:75–8.
14. Burkat CN, Lemke BN. Retrobulbar hemorrhage: inferolateral anterior orbitotomy for emergent management. Arch Ophthalmol. 2005;123:1260–2.
15. Ho VH, Wilson MW, Fleming JC, Haik BG. Retained intraorbital foreign bodies. Ophthal Plast Reconstr Surg. 2004;20:232–6.
16. Cho RI, Bakken HE, Reynolds ME, et al. Concomitant cranial and ocular combat injuries during Operation Iraqi Freedom. J Trauma. 2009;67:516–20.
17. Burnstine MA. Clinical recommendations for repair of orbital facial fractures. Curr Opin Ophthalmol. 2003;14:236–40.
18. Antonelli V, Cremonini AM, Campobassi A, et al. Traumatic encephalocele related to orbital roof fractures: report of six cases and literature review. Surg Neurol. 2002;57:117–25.
19. Cayli SR, Kocak A, Alkan A, et al. Intraorbital encephalocele: an important complication of orbital roof fractures in pediatric patients. Pediatr Neurosurg. 2003;39:240–5.
20. Ellis EE, Reddy L. Status of the internal orbital after reduction of zygomaticomaxillary complex fractures. J Oral Maxillofac Surg. 2004;62:275–83.
21. Markowitz BL, Manson PN, Sargent L, et al. Management of the medial canthal tendon in naso-ethmoid orbital fractures: the importance of the central fragment in classification and treatment. Plast Reconstr Surg. 1991;87:843–53.
22. Fattahi TT, Brandt MT, Jenkins WS, Steinberg B. Traumatic carotid-cavernous fistula: pathophysiology and treatment. J Craniofac Surg. 2003;14:240–6.
23. Savar A, Andreoli MT, Kloek CE, Andreoli CM. Enucleation for open globe injury. Am J Ophthalmol. 2009;147:595–600.
24. Kaltreider SA, Lucarelli MJ. A simple algorithm for selection of implant size for enucleation and evisceration: a prospective study. Ophthal Plast Reconstr Surg. 2002;18:336–41.
25. Migliori ME. Enucleation versus evisceration. Curr Opin Ophthalmol. 2002;13:298–302.
26. Bilyk JR. Enucleation, evisceration, and sympathetic ophthalmia. Curr Opin Ophthalmol. 2000;11:372–85.
27. Jordan DR, Stoica B. Evisceration with implant placement posterior to posterior sclera. Ophthal Plast Reconstr Surg. 2016;32:178–82.
28. Savitsky E, Eastridge BJ, editors. Combat casualty care: lessons learned from OEF and OIF. Fort Detrick: Borden Institute; 2012.

10 Neuro-Ophthalmic Manifestations of Trauma

Sarah J. Kim, Prem S. Subramanian, and Kimberly P. Cockerham

Traumatic Brain Injury and Optic Nerve Dysfunction

Traumatic brain injury (TBI) is an important cause of cognitive impairment and disability, imposing a burden on public health resources worldwide. The Centers for Disease Control and Prevention (CDC) define TBI as craniocerebral trauma occurring from an injury to the head from blunt, penetrating, or acceleration/deceleration forces [1]. TBI is a structural or physiological disruption of brain function accompanied by at least one of the following: loss of or decreased level of consciousness, amnesia, alteration in mental state (confusion, disorientation), neurologic defects (weakness, loss of balance, change in vision, praxis, paresis, sensory loss, aphasia), or intracranial lesion [1, 2]. Common causes of TBI in the civilian population include motor vehicle accidents, falls, assault, and sports injuries. It is estimated that in 2013, 2.8 million people in the USA sought emergency department care, were hospitalized, or died from TBI-related injuries [3].

The US Department of Defense (DoD) definition of TBI includes forces generated by a blast or explosion [4]. Combat blast is an increasingly important cause of military TBI, as exposure to powerful explosive devices is prevalent in current warfare and terrorism. Moreover, blast has become an increasingly common injury because of increased survivability from once-lethal blasts due to better personal armor, prompt casualty transport, and improved resuscitative and surgical care.

As of May 13, 2011, 43,300 US military personnel have been wounded in hostile actions in Operations Iraqi Freedom (OIF) and Enduring Freedom (OEF) since inception of combat actions on March 19, 2003 (www.defenselink.mil/news/casualty.pdf). Blast is the most common war injury [5] and largest cause of TBI, which is considered the "signature wound" of these conflicts [6]. More than half of US combat injuries result from blast-related trauma [7–9]. Blast accounted for 79% of injuries in a review of 1566 wounded soldiers in the Joint Theater Trauma Registry [5]. In a study in Veterans Health Administration, 84% of Polytrauma Rehabilitation Centers' inpatients and 90% of Polytrauma Network Sites' outpatients with moderate to severe TBI had blast exposure [10]. A report by the RAND Corporation in April 2008 estimated that 20% of all military personnel

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who have served in Iraq or Afghanistan (more than 300,000 individuals) may have some level of TBI due to blast exposure [8]. The actual number of US combatants in OIF and OEF exposed to blast is unknown.

Mechanism of Blast Injury

A blast explosion creates different mechanisms of injury via pressure, acoustic, electromagnetic, and thermal energies. Primary blast injury (PBI) is caused by barotrauma, due to overpressurization or underpressurization relative to atmospheric pressure. Secondary blast injury (SBI) occurs from fragmentation and projectiles causing direct blunt or penetrating injury to the individual. Tertiary blast injury is due to blast wind launching an individual against another object. Quaternary blast injury is from all other causes of injury, including burn, crush, and inhalation injuries.

In PBI, the blast wave is transmitted directly through tissues in two forms, stress waves and shear waves, which cause injury through different mechanisms [11, 12]. Stress waves of high frequency and low amplitude travel faster than sound in tissues. Energy is deposited wherever stress waves are reflected or change their speed, leading to a pressure differential between organs and the exterior. In other words, energy transfer and injury occur at the site of density change [13, 14]. Shear waves are transverse waves of long duration and low velocity which grossly distort tissues and organs and cause damage and disruption when imposed motion exceeds natural tissue elasticity. Wave reflections at interfaces of different density and conductivity create turbulence and cavitation, which dislocate fluid particles from denser to less dense tissue (spalling). As it travels through tissue, the blast overpressurization compresses gas, such as air within the gut, lungs sinuses, and middle ear, followed by rebound and a secondary shock effect. Indirect injury is produced by asynchronous motion of adjacent, connected structures with inertial differences, by stretching at sites of attachment, or by collision of tissues with more resistant structures, such as bone. These effects cause tears, contusions, and contrecoup injuries in soft tissues.

The velocity of the shock wave and the duration of the overpressure component are determined by three factors: the size of the explosive charge, the surrounding medium, and the distance from the explosion. Relatively small charges, such as hand grenades or small artillery, are designed to kill or disable by SBI, whereas larger charges kill and wound by PBI and deceleration, as well as SBI. IEDs placed in roadbeds and roadsides tend to produce blast close to targets (infantry or vehicles). Increased blast effect occurs in confined areas with reflecting surfaces [15], as in vehicles breached by an explosion. Position within the vehicle may also determine injury.

Armored vehicles, body armor, helmets, and ballistic eye protection are efforts to defeat the effects of blast and reduce injury. However, body armor, which is designed to stop projectiles, actually increases the effect of PBI on the thorax [16]. As use and capabilities of body and eye armor increase, insurgents strive to overcome these barriers by increased explosive force and proximity.

Traumatic Injuries to the Afferent System

Forces severe enough to cause TBI and associated polytrauma injuries may inflict injury to the lightly armored eye and adnexa. Reports suggest that sensory deficits, including visual complaints, are more common in TBI patients exposed to blast than from other causes of TBI [10, 17, 18]. Visual dysfunction may be a consequence of eye, visual pathway, or brain injury. Blast-induced ophthalmic damage may manifest as open globe injury, closed globe injury, orbital fractures, optic nerve injury, and dysfunctions of the visual and oculomotor systems.

Open Globe Injury

Open globe injuries due to rupture or penetration of the eye wall have been observed and reported over successive wars. Open globe injury may

occur from any of the mechanisms of blast injury, including from overpressurization, high-velocity projectiles, or from blunt forces such as impact with structures or debris. Open globe injury due to gunshot or fragmentation has been well understood by military healthcare planners, resulting in widespread use of polycarbonate protective eyewear in combat areas, training of frontline medics to recognize and triage eye injuries, and staffing of combat surgical hospitals with ophthalmologists capable of primary repair of eye wounds. In most cases open globe injury is apparent both to the casualty (if conscious), due to some degree of pain or visual loss, and to the caregiver by means of external inspection.

Closed Globe Injury

Injury to internal structures of the eye wall without rupture or penetration is classified as closed globe injury. Such injuries typically occur from blunt force mechanism, such as trauma from collisions, motor vehicle accidents, sports (baseball, tennis ball), or altercations (fist). Acceleration and deceleration forces secondary to an explosive source may also create blunt force trauma. Since the eye is a compliant surface, it will indent upon contact with a compressed shock wave from a nearby explosion. Directionality of the primary blast wave is likely to greatly influence the amount of ocular damage. Force is usually applied in an anterior-posterior direction, due to the protection afforded by the surrounding orbital bones. The orbital bones surrounding the globe will absorb some blast energy, while no such protection is afforded in the frontal plane.

Indentation of the eye leads to increased intraocular pressure and equatorial expansion due to incompressibility of intraocular fluids. Intraocular tissues are attached to the sclera, at fixed points. Expansion and pressure changes may shear or tear tissues at these fixed points. If sufficient loading is applied to the eye by mechanical trauma, the eye wall will rupture at its thinnest areas (usually at the limbus, or posterior to the extraocular muscle insertions) and become an open globe injury.

Closed globe injuries are often not apparent to an observer, unless blood in the anterior chamber (hyphema) is visible. A closed globe injury may go unnoticed by the casualty, as there may be no pain or obvious change in visual acuity [19], or the injury may occur in the context of decreased attentiveness or awareness due to head trauma. Closed globe injuries may lead to delayed vision loss through traumatic glaucoma or retinal detachment.

Closed globe injuries may affect any eye structure, including cornea (foreign bodies, contusion with corneal edema, tears in Descemet membrane, corneal endothelium damage); chamber angle (angle recession, cyclodialysis or ciliary body detachment, hyphema or blood in the anterior chamber, traumatic glaucoma); iris (iridodialysis, traumatic mydriasis); lens (opacification, traumatic cataract, subluxation); vitreous (vitreous hemorrhage, vitreous detachment); retina and choroid (choroidal rupture, retinal tear, retinal dialysis, retinal hemorrhage, traumatic retinopathy, retinal detachment); and optic nerve (contusion, ischemia, infarct, laceration, avulsion).

In addition to internal injuries described above from mechanical trauma, stress wave coupling and fluid-structure interaction create destructive internal forces within the confined space of the globe. Spalling at density interfaces between fluid vitreous or aqueous and more solid tissues, including corneal endothelium, iris, lens, optic nerve head, and retina, may cause tissue disruption. In a rat model, Petras et al. [20] demonstrated degeneration of the ipsilateral visual pathways, including optic nerve and optic tracts, after exposure to blast overpressure waves. They hypothesized retinal damage caused by the pressure wave.

Retrospective reviews of a population of inpatients with TBI from various causes at a VHA PRC have identified oculomotor dysfunction in a significant minority, including convergence and accommodative insufficiencies and impaired saccades and pursuits [21, 22].

Blast-Induced Traumatic Optic Neuropathy

While the majority of blast-induced ocular and orbital trauma is caused by SBI, it is important to be aware of the potential ophthalmic harm of PBI. With increased advances in gear protecting against incidence of SBI, ocular PBI may become an increasingly significant cause of vision loss and morbidity and may go unrecognized in a casualty with multiple injuries.

The optic nerve can be damaged directly or indirectly. Direct injury results from physical impact of the optic nerve or canal with an object, such as a foreign body or displaced bone from a fracture. Indirect injury results from an energy force originating elsewhere being transmitted and inflicting injury. The intracanalicular optic nerve is most vulnerable to indirect trauma, as forces transmitted anteriorly through the face and orbits tend to concentrate here. Damage within the optic canal is most likely ischemic, due to vascular compromise from the shearing of the nerve's pial nutrient vessels. Even minimal edema and hemorrhage of the nerve may cause additional compressive and ischemic injury, particularly within the tight confines of the optic canal. Shearing forces can also cause optic nerve avulsion. If the avulsion occurs anteriorly, a pit, or hemorrhage in a crescent or ring may be observed at the nerve head on ophthalmoscopy. The compromised retinal circulation may manifest as retinal hemorrhage or infarction. The fundus may appear normal if the avulsion is more posterior.

Intraorbital hemorrhage may lead to a compartment syndrome, causing compression and resultant ischemic necrosis to the optic nerve. Impact of the intracranial optic nerve against the falciform ligament can result in contusion necrosis.

While it is not completely understood how ocular tissues respond to blast injury, inflammation and apoptosis in optic nerves and retinal gliosis have been documented in rat models. Repeat exposure to additional head trauma or mild blasts causes more apoptosis. The damage is evident in the eye contralateral to the blast exposure, albeit to a lesser degree, and to bilateral optic tracts [23–25].

Diagnosis and Testing for Traumatic Optic Neuropathy

Traumatic optic neuropathy (TON) is a clinical diagnosis determined by careful history taking, examination, and imaging to rule out other causes of vision loss. Key signs of optic nerve injury include decreased visual acuity (color/brightness), visual field defects, and a relative afferent pupillary defect in unilateral optic nerve injuries.

If TON is suspected, CT imaging should be ordered to rule out other causes of vision loss, direct optic nerve injury, and fractures. CT imaging is also useful in identifying nerve compression and orbital hemorrhage. While MRI imaging may be a superior modality for soft tissue and optic chiasm viewing, a CT must be ordered first to rule out metallic foreign bodies.

Other testing that may be helpful but not necessary particularly in the acute setting, include visual field and optical coherence tomography (OCT). There is no pathognomonic visual field deficit in optic neuropathy, but various field deficit patterns may help in identifying the location of damage. Disruption of afferent pathways from the retina to cortex can be reflected in corresponding visual field defects. Reliable visual field testing, however, requires a patient with adequate vision, alertness, and cooperation. For unconscious or uncooperative patients, a visual evoked potential may be helpful. Noninvasive OCT imaging of the optic nerves can show active edema of the nerve head, or evidence of atrophy from previous damage.

Treatment for Traumatic Optic Neuropathy

Treatment may be feasible when there are axons that have survived an initial insult, and prevention of further damage from the effects of hemorrhage or edema is possible. Whereas optic nerve

injury due to transection or ischemia causes immediate permanent visual deficit, injury secondary to compressive forces may be amenable to prompt treatment. For orbital compartment syndrome due to hemorrhage, a lateral canthotomy and cantholysis should be performed immediately, if a ruptured globe can be ruled out. In cases of TON due to bone fragments from fracture or other direct cause, it is debatable whether surgical intervention will be of benefit from prompt removal of the impinging source, or if the damage is already done and irreversible.

Treatment of Indirect Traumatic Optic Neuropathy

There is no established treatment protocol for indirect TON (ITON). In some cases, improvement or spontaneous resolution of vision loss occurs in the absence of treatment. In the International Optic Nerve Trauma Study (IONTS), 57% of the untreated patients had significant improvement of three or more lines [26]. This same study did not find conclusive evidence of a significant benefit from steroid or surgical intervention. Conservative management with observation, therefore, is an acceptable approach.

Medical treatment typically consists of intravenous (IV) steroid therapy with methylprednisolone. There is still controversy about the use of IV steroid therapy in patients with head injury. The corticosteroid randomization after significant head injury (CRASH) study was cut short due to increased mortality with IV steroids in treating patients with acute TBI [27, 28]. For cases of ITON, low-dose IV steroid therapy has not shown significant visual benefit over conservative treatment [29]. Some studies show improved visual outcomes with high-dose IV steroids [30, 31], while some studies and most reviews do not find a significant difference compared to observation [32–34]. It is generally accepted that if used, high-dose IV steroids should be given within 8 hours of injury at a high dose of 30 mg/kg, followed by maintenance infusion of 5 .4 mg/kg/hour for 1–2 days in the absence of any contraindications (i.e., acute TBI). This regimen is extrapolated from studies of high-dose IV steroid treatment of acute spinal cord injuries, the National Acute Spinal Cord Injury Study I, II, and III [35–39].

Surgical treatment of ITON is primarily optic canal decompression via various methods: intracranial, endonasal, sublabial, and transethmoidal. Surgery alone can be a treatment option, as well as in conjunction with steroids, or after a failed treatment of steroids. Due to lack of comparative data, or any randomized controlled trials regarding surgical intervention for TON, it is widely perceived that there is debatable benefit of surgical intervention given its potential risks [40].

Other treatment options still under investigation include acupuncture, levodopa-carbidopa, transcorneal electrical stimulation (TES), and IV erythropoietin (EPO). A case series of 18 patients who received daily injections of 20,000 IU of EPO for 3 days, within 2 weeks of injury, showed significant improvement in best corrected visual acuity at 1 and 3 months. The authors propose that EPO has neuroprotective effects, reducing apoptosis, and neuroregenerative effects, promoting axonal growth [41]. Others have found similar positive visual outcomes with the use of EPO for cases of ITON [42].

Given a strong consensus in the literature toward conservative management of ITON, the options for treatment should be weighed on a case-by-case basis, and with the patient's informed consent with understanding of the risks, benefits, and alternatives available.

Ocular Motor Dysfunction and Diplopia

Adult-onset binocular diplopia may occur from a number of diseases affecting ocular alignment, most commonly related to dysfunction of cranial nerves (CN3, 4, 6) [43–45], or extraocular muscles (e.g., orbital trauma) [46]. In fact, trauma is among the most common cause of diplopia from any origin, potentially affecting cranial nerves, extraocular muscles, vergence, and supranuclear pathways controlling ocular alignment. In a large study of over 1100 patients treated for head injury

at a single institution, 79 out of 594 (11.6%) of patients with ocular manifestations of their trauma had cranial nerve palsy [47]. This population was quite young, and 70% of individuals were injured in road traffic accidents, and thus the group may closely resemble blast and otherwise injured soldiers. Analysis of traumatic cranial nerve injury in a more mixed population of inpatients and outpatients at a single institution with comparison of patients with and without cranial nerve palsy demonstrated that cranial nerve paresis was associated with worse short-term neurological outcomes. In addition, these patients had a lower Glasgow Coma Scale score in the acute setting, and they had a higher incidence of craniofacial injuries as seen on CT scan [48]. Traumatic third and sixth nerve palsies typically require a significant acceleration/deceleration injury, and their occurrence with seemingly minor trauma should prompt an investigation for an underlying structural lesion that may have predisposed the nerve to injury. In contrast, the fourth cranial nerve is much more vulnerable to injury because of its long intracranial course, and a fourth nerve paresis may occur after seemingly trivial trauma and without loss of consciousness. In military and civilian personnel with closed head trauma, transient binocular diplopia may occur in over 50% of subjects, while persistent double vision is seen in a much smaller population. Data from a comparative study of 500 military personnel with blast- (343) versus nonblast-related (157) mTBI suggest that persistent double vision is more common in nonblast injury, with 20% of the overall study population having a diagnosis of diplopia during initial evaluation and follow-up [49].

Diplopia often hinders a number of routine activities such as driving, reading, efficiently using the computer, and numerous other hand-eye-related tasks. The most common TBI-associated cause of diplopia appears to be convergence insufficiency. A retrospective analysis of 160 subjects with TBI and 60 after cerebrovascular accidents demonstrated that accommodative insufficiency was present in 41% of TBI subjects and only 12.5% of CVA patients, in whom saccadic deficits were much more common [50]. However, in other TBI patients, diplopic complaints may be more difficult to correlate with the presence of a measurable vergence disorder or strabismus during routine testing. Nonetheless, saccadic dysfunction in TBI patients, both civilian and military, has been recognized [51]. There is accumulating evidence that vestibular dysfunction may contribute to subtle abnormalities of ocular motor control [52], and its effect on saccadic control and accuracy has not been quantified fully. Higher cortical functions also drive these tasks; although the precise pathways may not be well delineated, the effects of even mTBI on saccadic speed and accuracy. Indeed, the emerging field of rapid concussion screening has developed methods for assessment of saccadic deficiencies that can be quantified by standardized reading tasks [53]. A limitation of such methods may be that baseline measurements are needed in order to assess subsequent dysfunction, as prior exposure to multiple head traumas can reduce long-term performance in the absence of an acute injury [54]. Furthermore, current diagnostic methods record reading speed as a presumed surrogate for saccadic accuracy and/or error generation. Pilot studies have assessed the eye movement errors and inaccuracies associated with reduced performance on rapid screening tests such as the King-Devick test, and they hold promise in helping us decipher which elements of saccadic control are preferentially impaired in mTBI [54]. Research and device development efforts are underway to create a standalone screener and eye tracker, likely through use of a virtual reality goggle system that incorporates position sensors for the globe as well. In the absence of these instruments, our methods remain an indirect assessment.

Despite the prevalence of ocular motor dysfunction in TBI patients in general and mTBI specifically, the majority of patients experience spontaneous and significant improvement in their symptoms after 6 months. No specific intervention has been demonstrated to be effective in hastening recovery or maximizing its likelihood. Methods that have been tried included part-time occlusion to relieve symptoms, base-in prism use

(especially in reading glasses), and orthoptic or optometrist-led visual training or vision therapy. Although ocular alignment and image separation may improve with additional time or following strabismus surgery in these patients [55], difficulties often persist and are difficult to characterize through standard clinical testing. Failure to capture these symptoms in objective testing may lead to a diagnosis of a functional disorder, which may not be appropriate. Other patients fail to benefit from interventions and remain diplopic with objective strabismus and are forced to occlude one eye, thus forfeiting stereovision (an important visual function that allows rapid depth perception used frequently in daily activities such as driving, walking, eating, etc.) [56]. This subset of patients may have damage to ocular motor and higher integrative pathways (as yet poorly delineated) that prevent them from reestablishing binocular fusion despite adequate treatment.

Testing and Treatment for Ocular Motor Dysfunction

As noted earlier, a number of treatments for diplopia are available, including prism therapy, strabismus surgery, and various forms of vision therapy including orthoptic training strategies [50]. These interventions often are reported in terms of resolution of diplopia in primary gaze and/or improvement in ocular alignment related to an arbitrary cutoff, but this fails to capture the broad view of the patient's visual experience. Diplopia is the end result of a complex interplay of several factors including not only the ocular motor injury but also fusional effort, time of day, distance, position of gaze, and head and body position. Patients also may over- or underreport their diplopic symptoms based on the intervention (or lack thereof) that they have chosen [10]. Neurologically impaired subjects have not been studied in a systematic way given the challenges posed by current testing methods and the inherent variability in measurements. Much of our knowledge to date comes from studies of military populations; an initial study of strabismic outcomes in soldiers (injured in Iraq or Afghanistan) with eye and/or neurological injury showed that they generally respond well to standard strabismus surgery, but diplopia status was not a study outcome [55].

While afferent visual disease (blur or loss of vision) is readily quantifiable using numerous well-studied and validated outcomes such as the Early Treatment of Diabetic Retinopathy Study or low contrast acuity charts, there are no standard quantifiable validated measures of efferent visual dysfunction to assess patients with diplopia or other binocular visual symptoms. The development of standardized afferent testing protocols was crucial in efforts to demonstrate the efficacy of interventions in chronic eye diseases such as diabetes and macular degeneration, and the methodology developed over 20 years ago remains the standard for outcomes in numerous clinical trials in ophthalmology and neurology. In contrast, the routine ophthalmic efferent visual exam includes subjective grading of ductions in each eye, and the more objective measures of ocular alignment such as prism and cover testing but lacks a standardized approach.

The absence of objective and reproducible measures of efferent visual disturbances has forced many practitioners to develop unvalidated, non-standardized methods that are not useful to the general ophthalmologist or optometrist in standard office practice. Data from small groups of patients screened with such methods also have been cited in an effort to introduce controversial treatment methods into the VA TBI care system [57]. Quantitative techniques to measure other TBI symptoms such as imbalance have shown promise in pilot studies and are being evaluated in larger populations to derive normative data in various brain-injured populations; diplopia requires validated outcome measures to allow efficient designs of clinical trials to evaluate potential treatments [58].

The patient's field of binocular single vision may be mapped using the Goldmann perimeter; when measured consistently, this provides a reproducible quantification of the area of single vision. However, the Goldmann test is done at near and thus fails to capture information about diplopia at distance and allows con-

vergence to influence the results (convergence influences the magnitude of both horizontal and vertical deviations). Additionally, the Goldmann's circular target is unable to acquire information about torsional diplopia, a symptom of primary importance because it fails to respond to prism correction and is common in post-traumatic CN4 palsy. Prism and cover testing require specific expertise and training, and inter-rater reliability is not ideal, with variability of up to ten prism diopters [59], making it difficult to know if subtle changes in the exam correlate with symptomatic changes. Hess screens can chart ocular rotations, but they do not measure binocular function, are not in widespread use, and fail to record torsional disparity. Similarly, many sophisticated eye movement tracking systems have been developed and are employed in oculomotor research labs, but the collection and interpretation of these data, which fail to capture binocular status, is a highly specialized activity beyond the ability of the general clinician. Since the tested subjects must maintain sustained attention during testing, the method also would be of limited utility in a TBI cohort.

Stereovision is a quantifiable assessment that incorporates binocular visual function in addition to visual acuity; while the presence of excellent stereopsis correlates with ocular alignment (fusion without diplopia) and intact afferent visual function (normal acuity), the interpretation of poor stereovision is more complicated. Patients may never have developed good stereopsis in childhood due to amblyopia or microstrabismus, but such patients do not necessarily have diplopia. An additional confounding factor is that adult patients with new-onset strabismus, who previously had good stereovision, may lose that acuity with prolonged and untreated diplopia (i.e., no periods of fusion). Decreased visual acuity (e.g., concomitant traumatic optic neuropathy or globe injury) also degrades stereovision. Nonetheless, the restoration of stereopsis comprises an objective endpoint in the evaluation of efferent disease, and in patients with recent onset of diplopia, the transient loss of fusion should be of lesser concern.

Prevention and Future Considerations

Since 2005, the use of ballistic spectacles or goggles has been mandatory for the US troops on combat missions in war zones. Ballistic lenses composed of polycarbonate are designed to defeat low-velocity projectiles, such as casing or debris liberated from fragmentation devices [59]. Proper use of ballistic eyewear will lessen the likelihood of open globe injuries in the combat zone, but little is known about performance of ballistic eyewear against blast pressure waves. Polycarbonate lenses may absorb some of the overpressure wave through deformation and breakage and thereby mitigate blast forces. However, experience indicates that body armor does not protect air-filled organs against barotraumas from the primary blast wave but may worsen the effect due to compression. Similarly, semi-rigid eyewear such as the currently available polycarbonate spectacles and goggles may be deformed and impelled against ocular and orbital tissues. Theoretically, this could increase compression of the globe and contribute to intraocular injuries.

Additional research is needed to understand the mechanisms of ocular tissue injury secondary to PBI, in order to develop corresponding methods of prevention. Further research is also necessary in developing protective eyewear capable of protecting against PBI in addition to SBI. Of equal importance is creating eyewear that does not compromise the wearer's visual clarity and field, in order to improve usage compliance.

References

1. Centers for Disease Control and Prevention, National Center for Injury Prevention and Control. In: Marr AL, Coronado VG, editors. Central nervous system injury surveillance data submission standards—2002. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control; 2004.
2. Corrigan JD, Selassie AW, Orman JA. The epidemiology of traumatic brain injury. J Head Trauma Rehabil. 2010;25(2):72–80.

3. Taylor CA, Bell JM, Breiding MJ, Xu L. Traumatic brain injury–related emergency department visits, hospitalizations, and deaths—United States, 2007 and 2013. MMWR Surveill Summ. 2017;66(SS-9):1–16.
4. Department of Defense. Memorandum on traumatic brain injury: updated definition and reporting. The Assistant Secretary of Defense, Health Affairs. 2015. Retrieved from: https://health.mil/Policies/2015/04/06/Traumatic-Brain-Injury-Updated-Definition-and-Reporting.
5. Owens BD, Kragh JF, Wenke JC, Macaitis J, Wade CE, Holcomb JB. Combat wounds in operation Iraqi Freedom and operation Enduring Freedom. J Trauma. 2008;64:295–9.
6. Menon DK, Schwab K, Wright DW, Mass AI. Position statement: definition of traumatic brain injury. Arch Phys Med Rehabil. 2010;91:1637–40.
7. Sayer NA, Chiros CE, Sigford B, Scott S, Clothier B, Pickett T, Lew HL. Characteristics and rehabilitation outcomes among patients with blast and other injuries sustained during the Global War on Terror. Arch Phys Med Rehabil. 2008;89:163–70.
8. Tanielian T, Jaycox LH, editors. Invisible wounds of war: psychological and cognitive injuries, their consequences and services to assist recovery. Santa Monica: RAND Corporation; 2008. Retrieved from: http://www.rand.org/content/dam/rand/pubs/monographs/2008/RAND_MG720.pdf.
9. Hoge CW, McGurk D, Thomas JL, Cox AL, Engel CC, Castro CA. Mild traumatic brain injury in U.S. soldiers returning from Iraq. N Engl J Med. 2008;358:453–63.
10. Brahm KD, Wilgenburg HM, Kirby J, Ingalla S, Chang C-Y, Goodrich GL. Visual impairment and dysfunction in combat-injured servicemembers with traumatic brain injury. Optom Vis Sci. 2009;86(7):817–25.
11. Phillips YY, Richmond DR. Primary blast injury and basic research. A brief history. In: Bellamy RF, Zajtchuk R, editors. Textbook of military medicine. Conventional warfare: ballistic, blast and burn injuries. Washington, DC: US Government Printing Office; 1989. p. 221–40.
12. Guy RJ, Glover MA, Cripps NP. The pathophysiology of primary blast injury and its implications for treatment. Part I: The thorax. J R Nav Med Serv. 1998;84:79–86.
13. Scott SG, Belanger HG, Vanderploeg RD, Massengale J, Scholten J. Mechanism-of-injury approach to evaluating patients with blast-related polytrauma. J Am Osteopath Assoc. 2006;106(5):265–70.
14. Wolf SJ, Bebarta VS, Bonnett CJ, Pons PT, Cantrill SV. Blast injuries. Lancet. 2009;374(9687):405–15.
15. Boffard K, MacFarlane C. Urban bomb blast injuries: patterns of injury and treatment. Surg Annu. 1993;25(Pt 1):29–47.
16. Cannon L. Behind armour blunt trauma—an emerging problem. J R Army Med Corps. 2001;147(1):87–96.
17. Goodrich GL, Kirby J, Cockerham G, Ingalla SP, Lew HL. Visual function in patients of a polytrauma rehabilitation center: a descriptive study. J Rehabil Res Dev. 2007;44:929–36.
18. Dougherty AL, MacGregor AJ, Han PP, Heltemes KJ, Galarneau MR. Visual dysfunction following blast-related traumatic brain injury from the battlefield. Brain Inj. 2011;25(1):8–13.
19. Cockerham GC. Blunt trauma and non-penetrating injuries of the anterior segment. In: Bellamy RF, Thach AB, editors. Textbook of military medicine. Ophthalmic care of the combat casualty. Washington, DC: Borden Institute; 2003. p. 137–48.
20. Petras JM, Baumann RA, Elsayed NM. Visual system degeneration induced by blast overpressure. Toxicology. 1997;121(1):41–9.
21. Magone MT, Cockerham GC, Shin SY. Visual dysfunction in combat related mild traumatic brain injury: a review. US Neurol. 2013;9(1):61–4.
22. Ciuffreda KJ, Kapoor N, Rutner D, Suchoff IB, Han ME, Craig S. Occurrence of oculomotor dysfunctions in acquired brain injury: a retrospective analysis. Optometry. 2007;78(4):155–61. https://doi.org/10.1016/j.optm.2006.11.011.
23. Choi JH, Greene WA, Johnson AJ, et al. Pathophysiology of blast-induced ocular trauma in rats after repeated exposure to low-level blast overpressure. Clin Exp Ophthalmol. 2015;43(3):239–46.
24. Wang HC, Choi JH, Greene WA, et al. Pathophysiology of blast-induced ocular trauma with apoptosis in the retina and optic nerve. Mil Med. 2014;179(8):34–40.
25. DeMar J, Sharrow K, Hill M, et al. Effects of primary blast overpressure on retina and optic tract in rats. Front Neurol. 2016;7:59.
26. Levin LA, Beck RW, Joseph MP, Seiff S, Kraker R. The treatment of traumatic optic neuropathy: the International Optic Nerve Trauma Study. Ophthalmology. 1999;106(7):1268–11277.
27. Roberts I, Yates D, Sandercock P, et al. Effect of intravenous corticosteroids on death within 14 days in 100008 adults with clinically significant head injury (MRC CRASH trial): randomized placebo-controlled trial. Lancet. 2004;364(9442):1321–8.
28. Edwards P, Arango M, Balica L, et al. Final results of MRC CRASH, a randomised placebo-controlled trial of intravenous corticosteroid in adults with head injury-outcomes at 6 months. Lancet. 2005;365(9475):1957–9.
29. Yip CC, Chng NW, Au Eong KG, et al. Low-dose intravenous methylprednisolone or conservative treatment in the management of traumatic optic neuropathy. Eur J Ophthalmol. 2002;12(4):309–14.
30. Chen HY, Tsai RK, Wang HZ. Intravenous methylprednisolone in treatment of traumatic optic neuropathy. Kaohsiung J Med Sci. 1998;14(9):577–83.
31. Pokharel S, Sherpa D, Shrestha R, et al. Visual outcome after treatment with high dose intravenous methylprednisolone in indirect traumatic optic neuropathy. J Nepal Health Res Counc. 2016;14(32):1–6.
32. Entezari M, Rajavi Z, Sedighi N, et al. High-dose intravenous methylprednisolone in recent traumatic optic neuropathy; a randomized double-masked

placebo-controlled clinical trial. Graefes Arch Clin Exp Ophthalmol. 2007;245(9):1267–71.
33. Yu-Wai-Man P, Griffiths PG. Steroids for traumatic optic neuropathy. Cochrane Database Syst Rev. 2011;19(1):CD006032.
34. Kumaran AM, Sundar G, Chye LT. Traumatic optic neuropathy: a review. Craniomaxillofac Trauma Reconstr. 2015;8(1):31–41.
35. Young W. NASCIS. National Acute Spinal Cord Injury Study. J Neurotrauma. 1990;7(3):113–4.
36. Bracken MB, Shepard MJ, Collins WF Jr, et al. Methylprednisolone or naloxone treatment after acute spinal cord injury: 1-year follow-up data. Results of the second National Acute Spinal Cord Injury Study. J Neurosurg. 1992;76(1):23–31.
37. Bracken MB, Shepard MJ, Collins WF Jr, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the second National Acute Spinal Cord Injury Study. N Engl J Med. 1990;322(20):1405–11.
38. Nesathurai S. Steroids and spinal cord injury: revisiting the NASCIS 2 and NASCIS 3 trials. J Trauma. 1998;45(6):1088–93.
39. Coleman WP, Benzel D, Cahill DW, et al. A critical appraisal of the reporting of the National Acute Spinal Cord Injury Studies (II and III) of methylprednisolone in acute spinal cord injury. J Spinal Disord. 2000;13(3):185–99.
40. Yu-Wai-Man P, Griffiths PG. Surgery for traumatic optic neuropathy. Cochrane Database Syst Rev. 2013;18(6):CD005024.
41. Entezari M, Esmaeili M, Yaseri M. A pilot study of the effect of intravenous erythropoietin on improvement of visual function in patients with recent indirect traumatic optic neuropathy. Graefes Arch Clin Exp Ophthalmol. 2014;252(8):1309–13.
42. Kashkouli MB, Pakdel F, Sanjari MS, Haghighi A, Nojomi M, Homaee MH, Heirati A. Erythropoietin: a novel treatment for traumatic optic neuropathy-a pilot study. Graefes Arch Clin Exp Ophthalmol. 2011;249(5):731–6.
43. Park U-C, Kim S-J, Hwang J-M, Yu YS. Clinical features and natural history of acquired third, fourth, and sixth cranial nerve palsy. Eye (Lond). 2008;22(5):691–6.
44. Rowe F, VIS Group UK. Prevalence of ocular motor cranial nerve palsy and associations following stroke. Eye (Lond). 2011;25(7):881–7.
45. Tamhankar MA, Biousse V, Ying G-S, Prasad S, Subramanian PS, Lee MS, et al. Isolated third, fourth, and sixth cranial nerve palsies from presumed microvascular versus other causes: a prospective study. Ophthalmology. 2013;120(11):2264–9. https://doi.org/10.1016/j.ophtha.2013.04.009.
46. Lyon DB, Newman SA. Evidence of direct damage to extraocular muscles as a cause of diplopia following orbital trauma. Ophthal Plast Reconstr Surg. 1989;5(2):81–91.
47. Sharma B, Gupta R, Anand R, Ingle R. Ocular manifestations of head injury and incidence of post-traumatic ocular motor nerve involvement in cases of head injury: a clinical review. Int Ophthalmol. 2014;34(4):893–900. https://doi.org/10.1007/s10792-014-9898-8.
48. Dhaliwal A, West AL, Trobe JD, Musch DC. Third, fourth, and sixth cranial nerve palsies following closed head injury. J Neuroophthalmol. 2006;26(1):4–10.
49. Capó-Aponte JE, Jorgensen-Wagers KL, Sosa JA, Walsh DV, Goodrich GL, Temme LA, Riggs DW. Visual dysfunctions at different stages after blast and non-blast mild traumatic brain injury. Optom Vis Sci. 2017;94(1):7–15.
50. Ciuffreda KJ, Rutner D, Kapoor N, Suchoff IB, Craig S, Han ME. Vision therapy for oculomotor dysfunctions in acquired brain injury: a retrospective analysis. Optometry. 2008;79(1):18–22.
51. Cifu DX, Wares JR, Hoke KW, Wetzel PA, Gitchel G, Carne W. Differential eye movements in mild traumatic brain injury versus normal controls. J Head Trauma Rehabil. 2015;30(1):21–8.
52. Kapoor N, Ciuffreda KJ. Vision disturbances following traumatic brain injury. Curr Treat Options Neurol. 2002;4(4):271–80.
53. Ventura RE, Balcer LJ, Galetta SL, Rucker JC. Ocular motor assessment in concussion: current status and future directions. J Neurol Sci. 2016;361:79–86.
54. Rizzo J-R, Hudson TE, Dai W, Desai N, Yousefi A, Palsana D, et al. Objectifying eye movements during rapid number naming: methodology for assessment of normative data for the King-Devick test. J Neurol Sci. 2016;362:232–9.
55. Subramanian PS, Birdsong RH. Surgical management of traumatic strabismus after combat-related injury. Mil Med. 2008;173(7):693–6.
56. Schaadt A-K, Schmidt L, Reinhart S, Adams M, Garbacenkaite R, Leonhardt E, et al. Perceptual relearning of binocular fusion and stereoacuity after brain injury. Neurorehabil Neural Repair. 2014;28(5):462–71. https://doi.org/10.1177/1545968313516870.
57. Padula WV, Ikeda E, Fong D, Vicci V. The need for optometric rehabilitation for our veterans who have incurred a traumatic brain injury: Senate Bill 1999/House Bill 3558. Optometry. 2008;79(4):170–1.
58. Wu-Chen WY, Christoff A, Subramanian PS, Eggenberger ER. Diplopia and quality of life. Ophthalmology. 2011;118(7):1481–1481.e2.
59. Hatt SR, Leske DA, Liebermann L, Holmes JM. Quantifying variability in the measurement of control in intermittent exotropia. J AAPOS. 2015;19(1):33–7.

Pediatric Ophthalmology

11

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Introduction

As innocent bystanders to the violence of war, children will not only be susceptible to and potential victims of all the combat traumas of adults, but they could also present with more routine noncombat conditions common to active children. US forces should be prepared to care for these children, as we may be their most definitive source of care. Trauma remains an important cause of ophthalmic pathology for the pediatric population both in a war zone and in the civilian sector. As a category, injury to the eye or its adnexa is the second leading indication for surgery among hospital-based pediatric ophthalmologists. Additionally, amblyopia associated with eye injuries, as a result of damage to ocular structures or prolonged postoperative visual deprivation, ranks as a significant cause of long-term vision loss. Ocular trauma is unfortunately a common occurrence in children, and victims often suffer substantial morbidity. While relatively few eye injuries require hospitalization and urgent surgery, the scope of the problem remains significant. Worldwide, it has been estimated that 8.85–15.2 per 100,000 children per year, or 160,000–280,000 children under 15 years of age sustain ocular trauma serious enough to require inpatient hospitalization [1]; 3.9 million people worldwide may suffer low vision in both eyes due to ocular trauma, while over 18 million live with unilateral visual impairment from trauma [1]. Trauma is thus the leading cause of monocular vision loss in young people worldwide.

In the United States, other estimates are as high as 840,000 eye injuries annually in the 18-and-younger population [2]. Most of these children are initially evaluated in the Emergency Department setting, and 95% of the injuries do not require hospitalization [1]. Indeed, many minor eye injuries will not come to the attention of the ophthalmologist. While the ultimate management of trauma in children is similar to that in adults in many respects, pediatric ocular trauma has many unique aspects we intend to define in the following pages.

Children can be difficult to examine, resulting in either a delay in diagnosis of a severe injury or, at the other extreme, exacerbation of an injury due to too forceful of an attempt to examine the unsedated patient. The history may not always be obvious, as the injury may not have been witnessed by a parent or adult. Fearing punishment, children will often be less than forthcoming

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about the cause or even existence of an injury, adding to the delay in appropriate treatment. Finally, unlike in adults, where good visual outcomes may occur months after the healing processes have taken their course, the dynamic nature of visual neurology in childhood makes for a challenge to the treating physician in the postoperative period. Rapid rehabilitation of the injured eye is required for binocularity to be reestablished and suppression, with resultant amblyopia avoided.

Epidemiology

In the United States, common household products are implicated in an estimated 125,000 pediatric eye injuries annually [3]. Sports and recreational activities are the most commonly implicated causes of eye injury, with 43% of ocular injuries involving sports occur in those 14 years and younger [3]. Baseball is the sport most responsible for eye injuries in children 14 and younger in the United States, while basketball is the most responsible sport in 15- to 24-year-olds [4]. Desk supplies (pens, pencils, etc.) are implicated more in 5- to 8-year-olds and toys in 2- to 4-year-olds, while falls and furniture-related accidents (walking into table corners, for instance) in those younger than 5 years [5]. Cleaning chemicals likewise cause more injuries in the less-than-five age group than other age groups [5]. During a 5-year retrospective analysis of eye injuries in Copenhagen, Denmark, boys between 6 and 15 years of age were most prone to eye trauma, and airsoft guns and fireworks were the most common injury etiologies [6]. In polytrauma victims with eye injuries, motor vehicle-related trauma, animal strikes, and falls are the most common etiologies [7].

Considering the larger population-based studies in the United States, [2, 5, 7] it is reasonable to think of children "9 and up" at the highest risk of ocular injury, while remembering that infants and toddlers are at higher risk of eye trauma from falls, furniture, and household chemicals.

While 95% ocular injuries do not require admission, in the United States, approximately 11,300 children with ocular injuries do require hospitalization annually, and of these injuries, 21–24% are penetrating globe injuries [1, 2, 7, 8]. An analysis of pediatric trauma victims from National Pediatric Trauma Registry stratified victims by trauma severity, with those with an injury severity scale (ISS) greater than 15 being classified as having "major trauma" [7]. Of interest, children with "major trauma" (ISS > 15) had fewer vision-threatening injuries (16%) versus those with ISS ≤ 15 (40%). Children w/ ISS scores >15 had more systemic injuries, orbital and facial fractures, and fewer isolated eye injuries.

All studies agree there is a male predominance to trauma. However, the overall proportions vary and range from 62.5% to 79.7% [1, 2, 5, 6, 9], with a higher male predominance when the causes of the injury are considered individually. This is not surprising; boys tend to be unaware or more naïve when it comes to possible dangers associated with certain activities, and when they are aware, they seem to be either foolhardy or fearless, in their approach, of the associated hazards. For example, air-gun injuries are found in a male-to-female ratio of 9:1, and fireworks 4:1–3:1 [1]. Desk supplies and cleaning products affect the genders more equally at 1.7:1 and 1.4:1, respectively [5].

History and Evaluation

Evaluation of the pediatric ocular trauma victim always begins with assurance that the child is systemically well enough to proceed with an eye examination. Airway, breathing, circulation, and neurologic status take precedent over suspected eye injuries. Any life-threatening injury should be addressed first. The initial provider's job is to evaluate the extent of ocular injury without causing further damage to the eye.

If a chemical burn is suspected, immediate copious irrigation with water, normal saline, lactated ringers, or BSS should be initiated, with care to irrigate the conjunctival fornices and remove any caustic sediment. Alkali products (e.g., drain cleaners, lye, bleach, ammonia, and

powdered detergents) are especially dangerous, given their ability to cause liquefactive necrosis and penetrate deep into the globe. Acidic compounds (vinegar, rust-removal agents, and metal polish) cause coagulative necrosis and substantial injury but are less likely to penetrate into the globe. Hydrofluoric acid (found in glass polish) is an exception and may behave more similar to an alkali agent on the eye [5]. Irrigation should be continued at least until the surface pH of the eye is neutral (6.8–7.4) [5]. If unsure about the properties of the exposure agent, contact poison control.

If open globe injury or eyelid laceration is suspected, CT scan, pain medications, antiemetics, and systemic antibiotics should be administered. Tetanus vaccination status should be verified or boosters given. Until an ophthalmologist can see the child, an eye shield should be placed to protect the eye, and efforts must be made to prevent Valsalva maneuvers, pressure on the globe, or other maneuvers that might extrude ocular contents. MRI is typically contraindicated in acute ocular trauma, and only an experienced ophthalmologist should perform ocular ultrasound (Table 11.1).

A history of trauma should emphasize the time frame of injury, the mechanism of injury, foreign body exposures, and a review of additional injuries. Symptoms such as pain w/ motility and diplopia may clue the provider of possible orbital trauma. Other medical conditions, prior surgeries, medications, allergies, and time of last meal should be recorded.

An accurate history can be challenging in pediatric trauma. In a nonverbal child, the caregiver may not have witnessed the trauma or may be unwilling to volunteer the circumstances surrounding the event. The ophthalmologist should always consider the possibility of abuse or neglect and contact the proper authorities if suspected. Verbal children, on the other hand, may be reluctant to admit to dangerous play and risk getting in trouble.

Table 11.1 Preophthalmic emergency department care of suspected open globes

C	CT scan
A	Antibiotics
T	Tetanus
S	Shield the eye

In any case, a history of a high-velocity, sharp, or explosive object should be concerning for penetrating or perforating open globe injury. Larger blunt objects may be concerning for orbital fractures, globe contusions, retinal injuries, or hyphema. Garcia et al. [7] noted that children with basilar skull fractures had an increased incidence of optic nerve injuries, an injury that might not be outwardly apparent. It was noted in the same study that many children had a delay in recognition of ocular injury in major trauma because they only appeared to outwardly have eyelid bruising or contusions compared to adults who more frequently had visible open wounds or communicated visual compromise. Pediatric victims of polytrauma with periorbital or facial fractures should thus all receive prompt ophthalmologic consultation.

In most cases, visual acuity is the strongest prognostic indicator in ocular trauma [10] and should be attempted in all children. In preverbal children, fix and follow behavior should be tested for each eye (using, for instance, brightly colored toys). HOTV match and the tumbling E can be done in cooperative children who are verbal but not literate, while Snellen acuity is best for older children. Many children with refractive errors will not have their spectacles available, so pinhole occluders and near-cards are a good substitute to assess best-corrected visual acuity. Motility, confrontational visual fields, pupil status, and a careful "front-to-back" exam then follow.

Children may need to be restrained or sedated to carry out a proper exam. If there is any suspicion of globe injury or the need for urgent surgery, an exam under anesthesia should be performed. Otherwise, parents and assistants may be needed.

Eyelid injuries may occur in combination with deeper injuries of the globe or orbit. An exam thus starts with inspection of the face and eyelids, looking for lacerations, foreign bodies, periorbital edema, ptosis, enophthalmos, or exophthalmos. Without magnification, inspect the globe for

foreign bodies, uveal prolapse, peaked pupil, and hemorrhagic chemosis. If significant lid swelling, Desmarres retractors or a speculum may be used to visualize the globe, with care to not apply any pressure to the globe. Eyelid lacerations should be examined carefully for injury to the canalicular system, and eyelids everted fully (e.g., with Desmarres retractors) to assess depth of injury. What may appear to be dirt on the surface of the globe can actually be iris or uveal tissue, so be careful before irrigating or brushing it away!

Orbital bone fractures are commonly encountered in children who suffer head and face trauma and have some different features compared to orbital fractures in adults. In all ages, blunt trauma to the globe or orbital rim may cause "blowout" fractures of the medial orbital wall and orbital floor. Unlike adults, however, children have elastic bones, and these fractures may form "trapdoors" that swing open with the initial force of trauma but then trap extraocular muscle and orbital fat as they swing closed when the tissue attempts to return to its original position. CT scan is sensitive for orbital fractures, but fractures may be clinically suspected by enophthalmos, ocular deviation and diplopia, and hypesthesia along the V2 distribution of the face. Extraocular muscle entrapment may be clinically apparent through severe pain w/ EOM, an occulocardiac reflex, and persistent nausea and vomiting [11]. If the child is sedated for exam, forced duction testing may provide valuable information about entrapment. Exophthalmos, on the other hand, may suggest retrobulbar hemorrhage.

Careful examination of the globe starts with the conjunctiva and sclera. Hemorrhagic chemosis is worrisome for globe rupture. Full-thickness scleral wounds may be distant from conjunctival wounds and are frequently difficult to see. Assess the cornea for any abrasions, lacerations, and punctures. Fluorescein dye is useful to assess for any active leak and should be used to look for this "Seidel Sign." If a self-sealing wound is suspected, careful probing after antisepsis is described, although controversial [12]. The anterior chamber depth can be easily assessed, and the presence of hyphema suggests injury to the iris and trabecular meshwork.

Once the globe has been cleared of visible perforation, eye pressure can be checked. An elevated eye pressure can be seen with hyphema, acute inflammation, lens dislocation, and retrobulbar hemorrhage, while low eye pressures may suggest occult globe perforation, cyclodialysis, or retinal detachment. Elevated IOP does not rule out open globe injuries [12]. Notable examination findings are listed in Table 11.2.

Patients with neurologic trauma frequently require serial neurologic exams with pupil checks. In these patients, careful documentation and communication with nursing and other

Table 11.2 A selection of ophthalmic findings in trauma

Face/eyelid	Canalicular damage
	Full thickness
Orbit	Orbital features
	Muscle entrapment
	Oculocardiac reflex
	Nausea/vomiting
Conjunctiva/sclera	"dirt" = uvea prolapse
	Hemorrhagic chemosis
	Laceration
Cornea	Abrasion
	Laceration/puncture
	Seidel sign
Anterior chamber	Shallowing
	Hyphema
	Inflammation
	Foreign body
Iris/angle	Peaked, irregular pupil
	Sphincter tears
	Iridodialysis
	Angle recession
	Cyclodialysis
Lens	Dislocation
	Vossius ring
	Cataract
	Capsular violation
Posterior segment	Vitreous hemorrhage
	Vitreous base avulsion
	Retinal hemorrhage
	Retinal tear
	Retinal detachment
	Retinodialysis
	Macular hole
	Commotio retinae
	Choroidal rupture
	Terson retinopathy
	Purtscher retinopathy
	Sclopetaria

specialties are important if a dilated funduscopic exam is acutely necessary. A full dilated exam should be performed at the time of injury, with attention to the lens for cataract, capsular damage, and lens dislocation. The vitreous is examined for pigment ("Shafer sign") or blood, and the retina for hemorrhage, tear, detachment, commotio, and other findings, but scleral depressed exam and gonioscopy should generally be delayed a few weeks with open globes or intraocular hemorrhage [12].

Ultrasound may be valuable in finding intraocular foreign bodies, retinal detachments, choroidal detachments, and posterior scleral ruptures in cases of poor posterior segment visualization. It requires an experienced ultrasonographer, however, as it is easy to inadvertently put pressure on the globe and extrude ocular contents [12]. Thus, it should generally be limited to experienced hands in controlled environments.

Common Injury Types

Of all children who present to the Emergency Room for eye trauma, corneal abrasions and contusions are the most common injuries, making up nearly half of ocular complaints. Conjunctivitis, chemical burn, foreign body, laceration, hemorrhage, thermal burn, and hematoma follow in that order [5, 13, 14]. Hyphema most frequently occurs after blunt ocular trauma, at an approximate rate of 8.7/100,000/year[1] to 17/100,000/year [15].

Of the children who suffered eye injuries in National Pediatric Trauma Registry [7], 35% of the injuries were categorized as vision threatening. The most common injuries were orbital wall fractures and contusions of the eye and adnexa, while injuries to the optic nerve and cranial nerves were relatively rare. Twenty percent of children with ocular injuries had basilar skull fractures, and of them, 27% had vision-threatening injuries. In children with major trauma, basilar skull fractures, orbital fractures, and optic nerve injuries were more common in major trauma. Open globe injuries were five times more common in those with less severe systemic trauma.

Common causes of eye injury are additionally discussed in the following sections.

Sports Injuries and Thrown Projectiles

Children suffer a very large number of eye injuries participating in sports every year, and it is estimated that 90% of these injuries are preventable. Children have shallower brows and flatter noses than adults, so they have less natural protection of their eyes. While it was once assumed that small projectiles were more apt to cause ocular trauma, every sport has been reported to cause eye trauma. Baseball seems to be the most associated sport in Americans younger than 14 years, while basketball is more associated in the 15- to 25-year-old group [4]. While the majority of injuries occur through projectiles (balls, shuttlecocks, flying disks, etc.), body collisions are implicated in nearly a third of cases [1, 16]. Blunt force injuries are most common, causing injuries such as hyphema, lens dislocation, commotio, retinal detachments, and globe rupture. It has been reported that 16% of sports-related eye injuries result in open globes [16, 17].

Of note, eye protection is uncommon in soccer, baseball, and basketball, while an eye-protecting helmet is mandated in American football, hockey, and lacrosse. Since Canada began mandating protective eyewear in ice hockey, ocular injuries declined more than 90% [16]. Field hockey-related eye injuries likewise declined 80% following the adoption of eye protection [18].

Stones and sticks were the most common etiologic agent for ocular trauma in Jamaica, usually in 4+ year-olds; 48.3% suffered contusions, while 35.9% had penetrating injuries [19]. Thrown eggs have been reported to cause substantial ocular injury, although open globe injuries have not been reported [1]. Snowballs generally do not cause many systemic injuries but are a common source of ocular injury. Most thrown projectiles occur in children older than 5 years [1].

Fireworks

Firework injuries are common worldwide, and over 5000 cases are seen in American children annually. Billock et al. [20] examined firework-related injuries in America between 1990 and 2014. Thirty percent of injuries were to the hands, while 21.5% were ocular injuries. Most ocular injures were in bystanders being struck by a firework or debris, rather than the actual explosion. Nearly half of eye injuries were diagnosed as corneal abrasions or burns, and the majority did not require hospitalization.

Bottle rockets have been documented to cause significant ocular injury. In a smaller American study of ten children [21], eight were boys, and all were launching the bottle rockets, but only one sustained injury to both eyes. Reported injuries included eyelid laceration, corneal abrasion, hyphema, iridodialysis, cataract, commotio retinae, traumatic maculopathy, and macular hole. Eight of the 11 eyes required urgent surgical intervention, and four required additional secondary surgery. While no patients had open globe injuries, final visual acuity was only better than 20/200 in four patients [21].

Nonpowder Guns

Nonpowder guns are an unfortunately common source of ocular trauma and cause substantial ocular morbidity, accounting for more eye injury-related hospitalizations than any other single source [15]. Airsoft guns use a spring mechanism to discharge lightweight 6 mm plastic beads, while BB guns shoot 4.5 mm steel or lead bullets, frequently using compressed air. Paintball guns use compressed air to shoot 17 mm paint-filled gel capsules. Projective velocity varies by launch mechanism (spring or air pressure) and ranges between 90 m/s in paintballs and 380 m/s in BB guns [15]. Because airsoft guns fire small plastic, lightweight pellets, they are frequently assumed to be a "safe" alternative to BB guns and air-rifles, and their popularity has thus exploded.

With the perception of their relative safety, airsoft gun-related play is frequently done without eye protection and causes more ocular injuries than other nonpowder guns, fireworks, etc. They generally cause ocular contusions without open globe injuries, while corneal abrasion, hyphema, iris trauma, cataract, and commotio are extremely common [6, 15, 22]. A small study of airsoft gun injuries found final visual acuity to be 20/200 or better in all patients, while only one achieved 20/40 [22]. Other studies find final visual acuity to be 20/40 or better in 65% [1].

Fig. 11.1 A 5-year-old male with phthisical eye resulting from a BB gun injury sustained at the age of 2 years

Traditional BB guns and paintball guns are more frequently associated with severe ocular trauma including open globe injuries, orbital penetration, retinal tear/detachment, choroidal rupture, and sclopetaria. Sclopetaria appears to be disproportionately prevalent after paintball injuries compared to other sources of trauma [1], while vitreous hemorrhage, hyphema, cataract, and retinal tears are commonly found [23]. Visual prognosis can be dire (Fig. 11.1) [1].

Chemical Injuries

Chemical injuries are most common in children younger than 5 years of age and typically happen at home [5, 13, 24]. Bilateral injury has been reported in approximately 18% of patients. Cleaning products and drain cleaners are particularly potent offenders and may be within reach of curious, unsupervised children. As stated above, alkaline agents cause liquefactive necrosis and penetrate through the cornea into the anterior chamber, while acidic compounds generally cause coagulative necrosis and affect only the surface (with the exception of hydrofluoric acid). Severe burns may require substantial surgical

management, and in the ambylogenic population, they typically affect and can be quite devastating. Worse prognosis is associated with delayed presentation. Prognosis varies, with an Indian study reporting visual acuity better than 20/50 in 12% of patients and worse than 20/400 in 64%.

Airbag-associated alkaline burns are also reported and may be associated with other ocular and systemic injuries [1]. Management can be particularly difficult.

Polytrauma

Ocular injury frequently occurs in victims of polytrauma due to motor vehicle accidents, motor vehicle versus pedestrian accidents, gunshot wounds, assaults, or animal attacks. An analysis of the National Pediatric Trauma Registry between 1987 and 2001 searched through ICD-9 diagnoses to find pediatric polytrauma victims who sustained eye injuries. Of the 96,879 patients included in the study, nearly two-thirds were male, had a mean age of 8.6 years, and 7.7% had eye injuries. In children with major trauma, vision-threatening injuries (globe lacerations/ruptures/contusions) were less prevalent in adults than in children classified as having less severe trauma (ISS $\leq$ 15). Children with ISS $\leq$ 15 suffered five times as many open globe injuries, while basilar skull fractures, orbital fractures, and optic nerve injuries were more common in major trauma [7].

Nonaccidental Trauma

Nonaccidental trauma (NAT), or *shaken baby syndrome*, should be uniquely considered as an injury type. Risk factors include low socioeconomic status, male sex, low birth weight, medical comorbidity, uncertain paternal identity, and social and family distress following natural disasters [1]. The exact incidence is difficult to quantify worldwide due to underreporting in many countries. The brain injury is more consequential to the patient's future vision than the retinal hemorrhages that characterize the condition [1]. See "Shaken Baby Syndrome" section of this chapter.

Animals

A fascinating review published in 2015 [25] discussed findings from 165 studies and series regarding animal-inflicted pediatric ocular trauma. A majority discuss insect or arachnid trauma, while domestic animal trauma, birds, and nondomestic mammals (in that order) make up most of the remainder.

Most reported cases of arachnid-inflicted trauma are from children (mostly boys) handling pet tarantulas. A tarantula defense mechanism includes shooting tiny hairs at the threat, and most of these cases involved keratitis, granulomatous uveitis ("ophthalmia nodosa"), and had a good prognosis after removal of the hairs and treatment with topical steroids and antibiotics. Brown Recluse bites made up the remainder of spider-related incidents and involved bites of the eyelid or adnexa, resulting in preseptal necrotizing cellulitis and, in one case, orbital cellulitis. Brown Recluse bites were thus associated with significant morbidity. Insect trauma from caterpillars most frequently involved caterpillars being handled or thrown and most often caused corneal erosion, keratitis, and/or uveitis (through caterpillar hairs being embedded in the globe, causing ophthalmia nodosa) with good visual outcome.

Bee stings have been reported, with lid swelling and keratitis the most frequently reported findings. Of particular interest, there have been at least seven documented cases [26, 27] of optic neuritis following bee stings to the face, including at least two children [27]. In most cases, the victim suffered acute visual acuity decline and bilateral optic nerve swelling following stings to the face. Improvement was rapid after IV steroids. The mechanism is thought to be due to demyelination secondary to the acute allergic reaction.

Fly larva ophthalmomyiasis (larvae being deposited in or around the globe) has been reported with a geographical predominance in

India and the United States. The lid is the most common site, followed closely by the ocular surface and, uncommonly, the globe. Reported cases of external ophthalmomyiasis had universally good visual prognoses, while internal ophthalmomyiasis resulted in LP or worse vision in 8 of 16 cases. The other 50% of cases had good vision [25].

The majority of bird-related injuries reported involved peck injuries from roosters and chickens, causing open globes [25]. Prognosis varied, but in one series of 14 bird-inflicted eye injuries, three cases were complicated by endophthalmitis [28]. In a more recent series of 30 patients in Iran [29], endophthalmitis developed in three patients with penetrating injuries.

Cats have been reported to be the cause of up to 2.8% of open globe injuries in children. An exception to the general rule may exist here as two-thirds of cases occurred in girls. Half of cases were complicated by *Pasteurella multocida* endophthalmitis [25]. Prognosis varies with the extent of injury.

Dogs are cited in up to 3% of ocular trauma cases requiring admission and in 1.4–3% of open globe injuries in children. Dogs frequently target the face for attack, and 9% of dog bites are found to affect the eye or periocular tissues. Among children with dog-inflicted eye injuries, there is a high rate of lid laceration and, particularly, canalicular injury.

Prevention

It has been frequently cited that 90% of eye injuries are preventable by the use of protective eyewear [30]. In sports, the adoption of mandated eye protection has drastically reduced the incidence of ocular injury in ice hockey, field hockey, and lacrosse [6, 7], while mandated eye protection certainly prevents innumerable eye injuries in those participating in paint ball. Where paintball is usually played in a controlled environment with eye protection, most eye injuries occur in unsupervised children. Unsupervised boys are particularly susceptible to injuring themselves, especially if they have access to dangerous toys (e.g., airsoft guns) and fireworks.

Simple steps to prevent such injuries can consist of locking up dangerous toys and releasing them only for play when supervised. For fireworks, BB guns, and airsoft guns, children under 10 years of age can be prohibited, and a "safety brief" with proper usage instruction, eye protection, and adult supervision may be instrumental in avoiding some injuries [21]. Other common household activities that may not seem particularly dangerous to the adult confer significant risk to the unprotected child. Weed-whackers and power lawnmowers may spray high-velocity debris into the eyes of foolhardy child operators or unsuspecting children nearby. Caregivers should provide appropriate role-model behavior by wearing eye protection in such activities, keep children a safe distance away, and enforce eye-protection use in child operators. The medical provider may support caregivers of children at risk of ocular injury with additional patient education and reinforcement of the dangers of injury.

Monocular children should always wear eye protection. Injury to the good eye can be devastating in these children. The ophthalmologist is obliged to discuss risky activity avoidance and eye protection use with the patient and family. Counseling is, however, often ineffective in convincing monocular children to wear eye protection and avoid risky activities. The ultimate responsibility of eye protection lies in the patient and family.

Fashionable eyewear, even plano spectacles, may encourage children who are otherwise resistant to wearing eye protection. Contact lenses should be discouraged in monocular children as they offer no protection and only add to the risk of eye infections. To be most effective, protective eyewear should be made of durable polycarbonate plastic and should fit snuggly. Glasses that continually slide down the nose or are easily displace with head movement (as in running or jumping) are inadequate as a suitable form of protection.

Orbital and Ocular Adnexal Trauma

Blows to the eye and orbit (Fig. 11.2) are a common reason for the pediatric patient to seek ophthalmic evaluation. Fractures of the roof, medial wall, and floor, all need to be treated acutely with

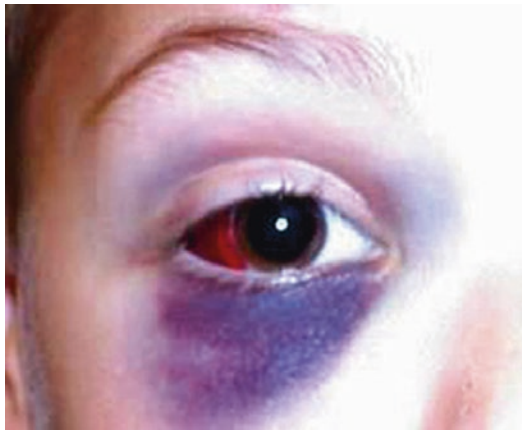

Fig. 11.2 Boy fell on his handlebars during a bike riding mishap

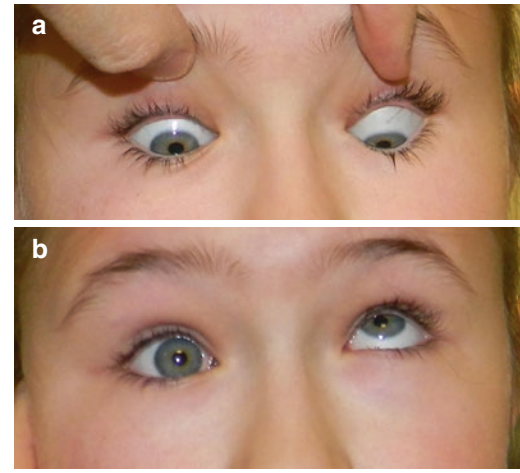

Fig. 11.3 Limited downgaze (**a**) and upgaze (**b**) of the right eye secondary to an orbital floor (blowout) fracture, with secondary entrapment of the right inferior rectus muscle

antibiotics to prevent orbital cellulitis, and patients should refrain from blowing their nose if possible. Orbital roof fractures, although rare in adults, are the most common type of orbital fractures in young children, aged 1–3 years, and are usually the result of forward falls, of only the height of the child, or similar limited elevation [31]. Nonaccidental trauma is not commonly associated with orbital roof fracture but should be considered if other signs of trauma of different ages and locations are noted. Significant edema and hematomas can result, almost always involving the upper eyelid. CT imaging is indicated, and a neurosurgeon should be consulted. Surgical repair is rarely needed. As the edema resolves, vision usually returns to normal quickly, but timely follow-up is required to confirm this. A few of these patients may develop partial levator dehiscence because of prolonged or severe edema and require a ptosis repair procedure later [32].

As with adults, orbital floor or medial wall fractures often result when a fist, knee, or ball strikes the socket or orbital rim. Unlike adults, the entrapment of the inferior rectus (Fig. 11.3a and b) may be more difficult to diagnose and/or treat, as the injury may seem less severe at the initial exam. Often the eye will appear quiet, without significant edema, without subconjunctival hemorrhage, and without the classic crescent hematoma of the lower lid. Some patients will quickly adopt a head position to avoid diplopia and thus may no longer have double vision as a complaint. A careful range of motion examination will usually reveal a significant restriction in upgaze and may also show some limitation of downgaze of the affected eye. This combination of findings is referred to as the "porcelain eye," "white eye," or "greenstick" blowout fracture and is a unique finding of the "trap-door" mechanism of injury commonly seen in childhood blowout fractures. CT scans are indicated, but up to 50% of these scans may not show the pathology in a definitive fashion. The trap-door fracture is frequently a fissure and not a true free bone fragment, and as the bone edges spring back into opposition, a portion of the inferior rectus can be entrapped. A history of a blow to the orbit followed by complaints of diplopia should make the examining ophthalmologist to maintain a high index of suspicion for this type of blowout fracture [32–34]. One quarter of these patients will present with nausea and vomiting, and some will have bradycardia or syncope, all of which are part of the occulocardiac reflex. These findings, in combination with the upgaze restriction, are reliable indicators for the need of surgical repair even in the event of a negative orbital CT scan.

Unlike the traditional 10- to 14-day waiting period for adult patients with orbital blowout fractures, surgery should not be delayed for

children, as the best results are obtained within 2–5 days of injury. Freeing the entrapped muscle is the goal, and an absorbable film or mesh style support can be used rather than a permanent type of orbital floor implant, as it is the entrapment and not the lack of orbital floor support that is the cause of the presenting symptoms [35]. In teens as in adults, significant enophthalmos and large size of the floor fracture can also be indications of surgical intervention. Transconjunctival incisions with or without lysis of the lateral canthus can give exposure to the fracture but sacrifice the lower lid retractor connections with the inferior rectus and rarely may result in injury to the inferior oblique muscle. The infraciliary approach produces a visible skin incision but does not violate the orbital septum and remains outside the orbital contents until the fracture site is encountered. These procedures are frequently done in cooperation with either ENT or maxillofacial surgeons. Medial wall fractures rarely need surgical intervention, for the diplopia associated with these injuries usually resolves with time as edema clears and the horizontal fusional amplitudes take over.

Long-term management often must address residual diplopia and include specialized strabismus surgery for noncomitant restrictive issues. The main goals are fusion in the primary position of gaze and in the reading position, with as great an area of single binocular vision as possible. Properly placed posterior fixation sutures of an unrestricted contralateral inferior or superior rectus yoke muscle can be helpful in slightly enlarging the field of single binocular vision.

Canalicular Tears

Children account for about 25% of all canalicular tears, with the clear majority of these tears due to dog bites. Usually, these injuries are avulsive in nature and involve the lower canaliculus almost exclusively. They are not directly due to the dog's canine teeth, which most frequently cause multiple punctures and superficial lacerations to the cheeks, lower lid, brow, and other parts of the face. Careful attention must be paid to these patients, as the injury may be subtle and hidden under blood and dried mucous. The caregiver may be easily distracted by more obvious lacerations and fail to consider the possibility of a canalicular tear. Once damage to the canalicular region is identified, successful repair is much more likely in the OR rather than at the ER bedside and should be done within 12–24 hours [36, 37]. The operating microscope is essential. The technique starts with punctal dilation and then probing of the involved lower lid canaliculus. The probe will appear at the torn distal end of the canaliculus. Identification of the proximal end of the torn canaliculus is best done by injecting a small amount of air into the superior canalicular system while leaving a pool of clear normal saline in the medial canthal region of the surgical field. Once identified, it is also probed, and then, a silicone stint, either mono-canalicular or bi-canalicular, is passed into the nasolacrimal duct. Two or three small sutures such as 9-0 nylon or 8-0 absorbable are used to reapproximate the torn ends of the canaliculus. The stint is removed usually 6–12 weeks later.

Anterior Segment Trauma

Corneal and Conjunctival Injury

The most common superficial eye injury in childhood is corneal abrasion. The history may indicate a mechanism, such as a patient's own fingernails or those of a parent or caregiver. In other cases, the patient may only present with symptoms of tearing, mild erythema, and voluntary eye closure or squinting of the affected eye. Diagnosis is facilitated with topical anesthetic drops, and frequently, once these drops are administered, the patient not only becomes easier to examine but also has a temporary reprieve from symptoms. Topical fluorescein staining and a blue-light source are used to confirm the diagnosis. Therapeutic pressure patching, the mainstay treatment of older children and adults, is often very difficult to achieve in young children. Topical antibiotic ointment such as erythromycin is usually all that is needed, and these abrasions

heal quickly but still require follow-up until fully healed [38].

Adolescents who are contact lens wearers suspected of a corneal abrasion must undergo a slit lamp exam to rule out the possibility of an undiagnosed contact lens-related bacterial keratitis. The erythema is usually more marked when keratitis is present, and there may be an associated anterior chamber cellular reaction. Even if no keratitis is detected, topical antibiotics, temporary suspension of contact lens use, and avoidance of an occlusive pressure patch are effective treatment options.

Chemical injury to the ocular surface is commonly seen in childhood, most frequently by household cleaning agents. Irrigation with tap water or bottled water immediately after the exposure has often already been performed by a parent or caregiver before seeking emergent care. By the time the child is examined by an ophthalmologist, there may be only a residual erythema accompanied by mild lid swelling in an otherwise asymptomatic patient. Vision almost always returns to baseline quickly. More severe chemical injury is associated with exposure to strong bases, such as drain de-clogging agents. In these cases, prolonged irrigation until normal pH can be obtained is the indicated treatment, followed by antibiotic ointment, cycloplegia, and topical steroids during the acute phase of therapy. Long term, these patients may require limbal stem cell transplant, amniotic membrane application, and even corneal transplant. Visual outcomes are guarded at best, especially during the age group at risk of amblyopia [39].

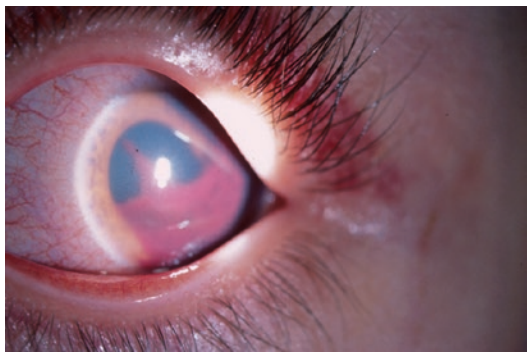

Fig. 11.4 Hyphema (blood in the anterior chamber) after blunt trauma to this child's left eye

Hyphema

Bleeding in the anterior chamber of the eye (Fig. 11.4), termed hyphema, is indicative of significant ocular trauma, either blunt or sharp, with damage and disruption to intraocular tissues. Two types of open globe injuries are lacerations and ruptures. Lacerations result from sharp objects entering (penetrating or perforating) the globe and ruptures result from blunt trauma causing a full-thickness defect at the weakest point of the eye wall. Blunt trauma to the eye, the most common cause of hyphema, often produces an indentation of the globe, especially in children younger than 3 years of age whose immature sclera is especially susceptible. This indentation can lead to stretching of the limbal tissues, equatorial scleral expansion, posterior displacement of the lens/iris diaphragm, iris sphincter tears, and acute elevation of intraocular pressure (IOP), with secondary tearing of the anterior face of the ciliary body and disruption of the major arterial circle and its branches [40–42]. Any of the aforementioned events can certainly lead to hyphema. Of course, if the history of a specific episode of trauma is questionable, one must consider other potential spontaneous, nontraumatic causes of intraocular hemorrhage including rarer etiologies such as retinoblastoma, juvenile xanthogranuloma, iris melanoma, hemophilia, myotonic dystrophy, leukemia, keratouveitis, von Willebrand disease, melanoma, rubeosis iridis [42, 43], or use of substances such as aspirin, coumadin, or warfarin that can negatively impact platelet or thrombin function.

In addition to measuring the visual acuity and performing a slit lamp examination, if possible, one should always check the hyphema patient's pupil reactivity and intraocular pressure (IOP) – especially in patients with a personal or family history of sickle cell anemia/trait. In these patients, specifically, the RBCs may become rigid, and if so, they are unable to easily traverse the trabecular meshwork possibly elevating the IOP – even with small hyphemas. Based on the slit lamp examination, Edwards and Layden [44] recommended the following grading system for hyphema: grade 1 – hyphema occupying less

than one-third of the anterior chamber; grade 2 – hyphema of greater than one-third but less than one-half of the anterior chamber; grade 3 – hyphema occupying one-half or more of the anterior chamber; and grade 4 – total hyphema (bright red blood) or eight-ball hyphema (dark red blood) filling the anterior chamber. The hyphema is considered microscopic (grade 0) when the red blood cells are seen circulating in the anterior chamber without layering. Gonioscopy should also be performed at some point to assess the angle structures for possible consequences of trauma, such as recession or scarring (peripheral anterior synechiae).

In terms of management, hospitalization of children with hyphema has both advantages and disadvantages. The advantages include ease of follow-up examination, medical compliance, and earlier diagnosis of complications [44]. However, the cost is the overriding disadvantage. Safe outpatient management depends on the family's and child's ability to comply with the prescribed activity restrictions, medication delivery, and scheduled reevaluation. Parents should be made aware of the seriousness of the injury and its potential complications (listed below). A firm agreement should be established with the parents and they should be instructed to keep their child's head of the bed elevated (30°–45°) to allow the hyphema to settle inferiorly out of the visual axis, significantly limit the child's activity, insist the child wear an eye shield at all times, and, at least initially, follow-up daily.

Medical management is time-tested and consists of topical steroids (prednisolone acetate 1% every 2 hours) and topical cycloplegia (atropine 1% twice daily) – taper gradually. In the event of increased IOP, topical aqueous suppressants (beta-blockers or alpha-agonists) should be employed initially. If topical medicines prove inadequate for IOP control, systemic medicines such as carbonic anhydrase inhibitors and/or hyperosmotic agents such as mannitol or acetazolamide – both of which are to be avoided in patients with sickle cell disease, SC disease, or sickle trait – may be required to achieve IOP control. If all of the above fail to control the IOP, surgical intervention may be required.

In an attempt to lessen the likelihood of a rebleed in hyphema patients, treatment with antifibrinolytic agents, aminocaproic acid or tranexamic acid, helps to prevent clot lysis, giving the injured vessel(s) time to recover from the initial insult. Tranexamic acid is more potent and has fewer side effects than aminocaproic acid. Although neither agent is widely used in children, they could both be beneficial in patients at high risk for a re-bleed or complications such as glaucoma, corneal blood staining, pupillary block, posterior synechiae, peripheral anterior synechiae, and amblyopia.

Fortunately, in this author's experience, very few patients (~5%) with hyphema require surgical intervention. After an adequate trial of maximal medical treatment, indications for surgery include a hyphema that persists in the face of increased IOP (greater than 50 mmHg for 5 days or more, greater than 35 mmHg for 7 days, and a large or total hyphema with an IOP greater than 25 mmHg for 5 days or over 24 hours in sicklers) [40, 42], corneal blood staining, residual or increasing hyphema, or a hyphema so large that it obstructs the visual axis of young children.

The simplest surgical option consists of irrigation and aspiration (washout) through a small limbal incision. If a significant clot is present in the anterior chamber, anterior vitrectomy instrumentation may be utilized. With persistently elevated intraocular pressures, a trabeculotomy may be indicated. The prognosis is dependent on the cause and associated complications, if any, of the hyphema. After a single bleed, the visual prognosis is excellent, with 91% achieving 20/30 or better vision. The visual prognosis is more guarded with a secondary hemorrhage, with only 77% having 20/30 or better vision. Amblyopia in patients under the age of 9 years and traumatic cataract are infrequent complications [44, 45].

Traumatic Cataracts

In a retrospective series of traumatic cataracts in children reported by Reddy et al. [46], approximately half of the 25 cases of traumatic cataracts reviewed were caused by nonperforating injuries.

BB and paintball injuries were by far the most common cause of cataracts by blunt or concussive injury, whereas pens and pencils were the most common cause of penetrating and perforating injuries. They reported that ocular injuries resulting from paintball and BB pellet impact are often so severe that treatment is sometimes limited to an attempt to salvage what remains of useful vision [47, 48]. In Reddy's study, 75% of children younger than 6 years required treatment for amblyopia and had a poorer visual outcome, suggesting that amblyopia may be a limiting factor for visual rehabilitation.

Whether traumatic cataract is caused by a perforating (Fig. 11.5) or nonperforating injury, management is dependent on the age of the patient, the amount of corneal damage, and the amount of remaining capsular support. The first priority in either case is to restore/maintain the integrity of the globe and clear the visual axis. Corneal pathology (scarring), cataracts, and optical needs must be promptly addressed in order to permit injured children to regain, continue to develop, and maintain "good" vision. In unilateral cases, these sorts of injuries put the affected eye at a distinct disadvantage, and for those victims under the age of 8 years, overlying amblyopia may complicate the visual rehabilitative process. Ophthalmologists must team with parents to ensure that they realize their role is vital in their child's visual rehabilitation.

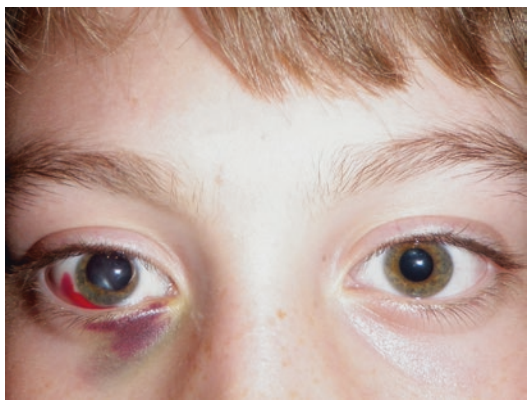

Fig. 11.5 Spine of fish tossed by his father struck this child's right eye causing lid ecchymosis, a subconjunctival hemorrhage, corneal perforation, and sub-total traumatic cataract

In adults and children beyond the age of 8–10 years when amblyopia is no longer a major concern, the approach to combined cornea–lens trauma is more straightforward. The older patient's globe integrity is initially reestablished, and once healed, the traumatic cataract is secondarily removed and an IOL is implanted. Calculating the intraocular lens (IOL) power in a healed adult eye is predictably more accurate and technically easy. Children, however, have the overlying and visually significant issue of amblyopia necessitating rapid intervention and the consideration of an urgent triple procedure (corneal transplant, cataract extraction, and IOL implantation). One must weigh the advantages of early visual rehabilitation against potential complicating factors such as severe postoperative inflammation, management of the posterior capsule, the high risk of corneal graft rejection, and antiamblyopia therapy [49].

Pediatric aphakia can be treated in a variety of ways – spectacles, contact lenses, or intraocular lenses. The majority of traumatic cataracts are unilateral, and in children with unilateral aphakia from whatever the cause, spectacle correction is suboptimal due to anisometropia, aniseikonia, and induced prismatic effect of the single aphakic spectacle lens. Contact lens rehabilitation presents its own set of problems. If the cornea is traumatized, sutures and/or scarring may make contact lenses unfeasible. Moreover, parental compliance with an uncooperative child is another serious consideration, not to mention the expense associated with frequent contact lens loss. Therefore, many surgeons opt for initially reestablishing the integrity of the open globe and returning at a later date for secondary IOL implantation, believing that with a stabilized surgical wound and reduced inflammation, surgery in a "quiet" eye that often results in better outcomes. According to the Pediatric Keratoplasty Association [49], however, if a combined penetrating keratoplasty, cataract extraction, and IOL implantation could be performed safely, with reasonable refractive accuracy and a high rate of graft clarity, severe amblyopia could be avoided and should be considered the procedure of choice in children with this type of trauma.

With adequate capsular support, the implantation of an IOL into the capsular bag or ciliary sulcus is fairly straightforward. However, trauma-induced compromise of the capsular bag and/or zonules makes IOL implantation much more challenging. Traditional techniques such as implantation of anterior chamber IOLs have less favorable outcomes because of frequent complications such as corneal decompensation and secondary glaucoma [50, 51]. There are conflicting reports regarding whether scleral-fixated IOLs are better than open-loop AC IOLs in terms of long-term visual outcomes and complication rates [52, 53]. Conventionally, scleral-fixated IOLs have been sutured to the sclera using double-armed 10-0 nylon polypropylene sutures; however, this technique is cumbersome and takes considerable time and effort to perform [50]. 10-0 polypropylene suture can degrade after 7–10 years, resulting in spontaneous subluxation of the IOL. This presents a particularly important concern for pediatric patients because their life expectancy is typically measured in decades and they are generally much more active than older adults. The constant physical stress that an active lifestyle exerts may accelerate the breakage of the suture material [53]. Price et al. [54] and Stewart and Landers [55] recommended that 9-0 polypropylene suture be used as an alternative to 10-0 polypropylene because it has a 60% greater tensile strength, 50% greater diameter, and a 125% greater cross-sectional area. The only drawback of using this suture is the size of the knot, which is larger and requires an even more concerted effort to adequately protect it from eroding through the conjunctiva [56].

Sutureless fixation of a scleral-fixated IOL was first described by Gabor and Pavlidis [57] and includes implantation of a three-piece IOL with exteriorization of the haptics through a scleral opening. The haptics are then buried inside specially created scleral pockets. This technique is said to be technically less demanding and quicker to perform, thus easier to master [50]. Reported complications associated with this technique include haptic erosion and degeneration, pupillary capture, retinal tears and detachments, cystoid macular edema, hypotony, and endophthalmitis [58]. Synder and Perez [59] described an alternative technique for sutured scleral-fixated IOLs using polytetrafluoroethylene (Gore-Tex) with a girth hitch for IOL fixation, achieving what they describe as better centration and the avoidance of torque. In a comparative study, Sindal et al. [50] determined that initial judicious surgical care in cases of trauma, with scleral-fixated IOL implantation at a later date, can give excellent visual outcomes. They determined that whichever surgical technique is utilized, the ultimate visual outcome is dependent on the underlying ocular pathology, such as corneal or retinal scarring. Moreover, this group found no differences in outcomes and complication rates between those having sutured scleral-fixated IOL implantation and those having sutureless scleral-fixated IOL implantation.

Greenwald and Glaser [60] compared subgroups of children with traumatic and nontraumatic cataract in whom the preoperative visual acuity was <20/100 and reported that PCIOL implantation seemed to provide significantly better binocular function but did not substantially improve the visual acuity results. Their recommendations to achieve the best visual results in children with traumatized eyes include prompt extraction of traumatic cataract with either primary or secondary IOL implantation, opening of any posterior capsular opacification either surgically as part of the primary repair or secondarily with Nd:YAG laser, correction of refractive errors, and vigorous anti-amblyopia treatment. Of course, surgical central posterior capsulotomy leaving sufficient capsular support for future IOL implantation combined with an anterior core vitrectomy at the time of cataract extraction in an injured child under the age of 2 years is this author's preference.

Posterior Segment Trauma

Vitreous Hemorrhage (VH)

Dana et al. [61] reported that in patients older than 10 years, the common causes of vitreous hemorrhage (VH) were proliferative diabetic ret-

inopathy (35.2%), trauma (18.3%), vein occlusion (7.4%), retinal tear without detachment (7.0%), posterior vitreous detachment (6.5%), proliferative sickle retinopathy (5.7%), and retinal tear with detachment (4.8%). In 4% of their patients under 10 years, trauma was the sole cause of vitreous hemorrhage. In a study of children under the age of 18 years, Spirn et al. [62] identified 26 different causes of VH in their population. Penetrating and nonpenetrating traumas accounted for the vast majority of cases (54.3% combined). When combined with occult causes of trauma (nonaccidental trauma and birth trauma) and postoperative patients, all trauma accounted for 75% of cases. Spontaneous causes made up approximately 25% of cases, with regressed retinopathy of prematurity (ROP) being the most frequent (5.9%). Familial exudative vitreoretinopathy (FEVR) and pars planitis were two other relatively frequent causes of VH. Bilateral VHs occurred in almost 10% of the study population. Nine different etiologies accounted for the bilateral hemorrhages, with shaken baby (impact) syndrome comprising 50% of bilateral cases and 8.6% of all cases.

In Spirn's series, children older than 9 years of age with vitreous hemorrhage presented with decreased vision, floaters, and pain. In contrast, the youngest children (<3 years) most frequently presented with strabismus, nystagmus, abnormal pupillary reflex, or behavioral change. Visual outcomes were poor, with a mean final VA of 20/277 for patients able to read the Snellen charts with 3 months' follow-up. Severe visual loss (20/800 or worse) occurred in 40 of 103 eyes. In terms of management, Sebag [63] suggested a 2- to 3-week delay of vitrectomy is acceptable with vitreous hemorrhage without associated retinal detachment. However, if extensive and visually significant, consideration of delay should be weighed against the hemorrhage's amblyogenic effects. If amblyogenic, pars plana vitrectomy (PPV) should be considered within 14 days of injury – especially if zone 2 and/or 3 is involved – to prevent tractional RD [63]. Outcomes in children with VH were generally poor and appear to be more dependent on etiology than age. Regressed ROP had a mean final VA of 20/31, whereas penetrating trauma had a mean final VA of 20/582 in this study.

Retinal Detachment (RD)

The epidemiologic features, anatomic characteristics, and prognosis of rhegmatogenous retinal detachment (RRD) in children differ from the detachment in adults. Manshot [64] classified causes of retinal detachment in children as follows: developmental, retrolental fibroplasia (ROP), trauma, inflammation, Coat disease, and retinal tumor. Hudson [65] reported 35 retinal detachments in children younger than 15 years of age. Fifty-one percent had a history of trauma. Tasman reported 52 patients between 3 and 16 years of age with RRD, of which 28.8% had a history of trauma, 21.2% had a history of prematurity, 15.4% had undergone lens aspiration for congenital cataract or ectopia lentis, and 3.8% had a history of uveitis [66]. Okinami et al. [67] reviewed a very large series of children (908 eyes) younger than 19 years treated for RRD in Japan. However, only 7% of these were in children 9 years of age or younger. Trauma was thought to be a factor in 26.4% of all eyes.

In a study published in 2014 out of Saudi Arabia, AL-Zaaidi, et al. [68] reported their experience with rhegmatogenous retinal detachment (RRD) in children 16 years of age or younger. The incidence of RRD in children is low, accounting for approximately 1.7–5.9% of all retinal detachments [69–74]. There is a greater preponderance of pediatric retinal detachments in males (70–79% of all cases), and the most common retinal break is a horse-shoe tear [74]. Etiologic factors include congenital or structural abnormalities, blunt or penetrating ocular trauma, myopia, a positive family history, and previous ophthalmic surgery. Retinal injury is known to occur during surgical procedures for congenital cataract, cryotherapy, laser photocoagulation in retinoblastoma, retinopathy of prematurity, and other procedures [72]. Kiffney [75] was the first to report retinal detachment secondary to child abuse.

The AL-Zaaidi group found the majority of fellow eyes (60%) were also affected and could be treated early. Seventy percent of their patients were male. The most common presentations were a decrease or loss of vision (45.7%) and incidental findings on routine exam (23%). A total of 86.75% of their patients had retinal breaks or holes. Forty-three percent had a history of trauma, and more than half (50.6%) had undergone previous ophthalmic surgery. Thirty-one percent had developmental or ophthalmic/systemic associations with RRD and 33% were myopic. Of the retinal breaks, 62% cases were holes or tears, 20.8% were retinal dialyses, and 22.9% were giant tears. In their study, pars plana vitrectomy with or without a buckling procedure was the primary surgical procedure in 85% of their cases. The success rate after the first surgery was 63.8%, and the final retinal attachment rate was 80.4%. They determined predictors of recurrence were myopia, PVR, and total RRD. In their study, predictors of good final VA were an initial VA of >20/200, absence of PVR, and attached macula at presentation. Negative predictors of final VA were development of complications after the first surgery (recurrent RD), postoperative PVR, epiretinal membrane, and ruptured globe repair. PVR incidence in children is 29.8–37.5%, while in adults, it is 5–10% and may be due to delayed diagnosis [68].

The ocular trauma scale (OTS) takes into account the visual acuity at presentation, presence of rupture, penetration, retinal detachment, endophthalmitis, and afferent pupillary defect but does not consider other factors such as macular attachment and duration of macular detachment, which may certainly influence the final visual acuity. Sarrazin et al. [69] found the only predictor for visual acuity of 20/200 or better was macular attachment. In a separate study, Winslow and Tasman [70] noted that retinal dialysis with vitreous base avulsion is typical of closed globe injury but was infrequently encountered. They added that in addition to direct retinal insults by penetrating objects, the similarities in the characteristics of retinal detachment after open and closed globe injuries suggest that both trauma types potentially cause secondary indirect impact injuries that cause deformation of the globe. They reported factors having particularly poor prognostic value include total retinal detachment, fixed retinal folds, proliferative vitreoretinopathy, large dialysis (>90°), multiple retinal tears, posterior tears, missed retinal breaks, and giant retinal tears. Additional factors contributing to poor outcomes in posterior segment trauma were late surgery; severe vitreous hemorrhage; poor visual acuity at presentation; large lacerations; blunt, missile, and BB injuries; presence of retinal detachment; and younger age [71–77].

Open Globe Injury

Open globe injuries (OGI) can be categorized by zone of injury. Zone 1 includes the cornea (Fig. 11.6) and limbus. Zone 2 extends from the limbus to the anterior 5 mm of sclera, and Zone 3 extends posterior to Zone 2 [78]. When assessing a cooperative child with an open globe injury, be aware of the correlation of reported factors with good prognosis and visual outcome, which include good initial vision, injury caused by sharp objects, corneal and/or scleral laceration of relatively short length, injury anterior to the rectus muscle insertions, and the absence of an afferent pupillary defect, lens injury, or hyphema [79–82]. However, one must be cognizant of the fact that many children, especially victims of significant ocular trauma, are often very anxious and unable to cooperate with the visual acuity assessment or a reliable pupil examination, and therefore, those aspects of the initial encounter cannot be relied upon to have the prognostic significance they have in older patients. Open as opposed to closed globe injuries in children are generally more severe, and a detailed examination under anesthesia is often required to fully determine the extent of such injuries.

When an open globe is suspected either domestically or on the battlefield, first, responders should apply a rigid shield over the injured eye after the initial assessment. Do not apply a patch that might add pressure to the globe or

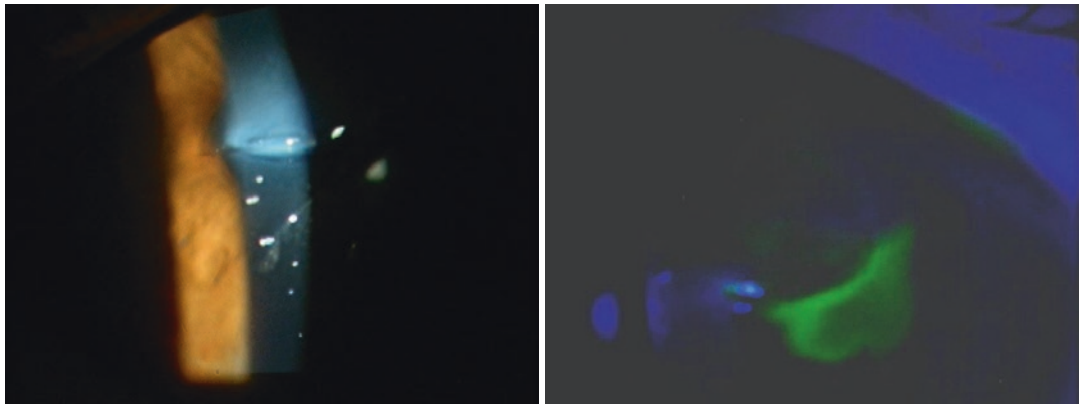

Fig. 11.6 Corneal laceration with leaking aqueous humor confirmed by positive Seidel's test

adhere to the already extruded intraocular contents potentially compounding the severity of the original injury. In addition to a shield, antibiotics (such as a first-generation cephalosporin such as cefazolin, or if an IOFB is suspected, a course of antibiotics such as intravenous or even intravitreal vancomycin and ceftazidime should be considered but have not been widely tested or accepted in children), analgesics, and possibly antiemetics should be administered. If the immunization status of the victim is at doubt, a tetanus shot should be given as well.

An intraocular foreign body (IOFB) should be suspected with a history of explosion, gunshot wound, or sharp object entering the eye, and appropriate imaging should be ordered [83–86]. Either computerized tomography (CT), gentle B-scan ultrasonography by an ophthalmologist or highly skilled ophthalmic technician, plain film X-ray, and/or magnetic resonance imaging (MRI) should be considered. The CT scan is highly sensitive for detection of IOFBs – especially metallic ones – less so for glass; both appear hyperdense. Wood, on the other hand, appears hypodense. As a rule, in the face of an open globe, B-scan ultrasound or any diagnostic technique that adds pressure to an open globe should be avoided if at all possible. Plain films (X-rays) are valuable for detecting IOFBs and bone fractures. MRIs are only useful for detecting nonmetallic IOFBs. Ferromagnetic material (e.g., nickel, iron, or cobalt) will be displaced by the magnets in the MRI, whereas other metals (e.g., tantalum) will not [1, 87–90]. Whether the history is significant for an intraocular body or not, a high clinical suspicion for occult scleral laceration should exist in the setting of hemorrhagic chemosis, hypotony (intraocular pressure (IOP) < 10 mmHg), an abnormally deep or shallow anterior chamber, or a peaked pupil [1].

Sympathetic ophthalmia is rare. Its incidence after trauma is reported as less than 1% in pediatric patients [91]. If diagnosed or even considered, high-dose corticosteroids followed by steroid-sparing agents and/or immunosuppressant agents in refractory cases should be considered. Long-term, chronic inflammation may be a sign of a retained, previously undetected IOFB. Metallic IOFBs are typically the most toxic [92]. Copper IOFB toxicity is called chalcosis bulbi. Any IOFB with a copper content of 85% or more can produce an intense inflammatory response including hypopyon and may present as a sterile fulminant endophthalmitis. IOFBs with a lower copper content can lead to a greenish discoloration of Descemet membrane, a reddish-brown cataract and uveitis progressing to phthisis [93]. Siderosis bulbi is the term applied to the toxicity caused by retained iron IOFBs, which can induce retinal degeneration with progressive visual loss [94] and retinal vascular change. Mydriasis is an

early sign progressing to rapidly increasing IOP. Decreased B waves are seen on electroretinogram [95, 96].

Trauma is also one of the most common causes of endophthalmitis in children [97]. Risk factors include the presence of an IOFB, injury in a rural setting, wound contamination with organic matter [97, 98], primary wound closure delayed for longer than 24 hours post-injury, and involvement of the lens capsule. Consider gram-positive normal skin flora such as *Staphylococcus epidermidis*, or more virulent species such as *Bacillus* sp. and *Streptococcus* (rarer in children than in adults). If the injury is caused by contact with vegetative matter such as a tree branch, fungal infections such be considered. Moreover, very virulent organisms such as *Bacillus* sp. and *Clostridium* sp. should be considered if the child has been in contact with soil.

Ocular Complications of Head Trauma

Traumatic Cranial Nerve Injury

Closed head trauma or, rarely, sharp trauma may cause partial or complete palsy to CN 4, CN 6, and, very rarely, CN 3. Similar surgical techniques are useful for CN palsies, to include, inferior oblique weakening in cases of CN4 (trochlear) palsy and generous ipsilateral medial rectus recessions for partial CN6 (abducens) palsies, with or without a posterior fixation suture of the contralateral medial rectus. Complete CN6 palsies require a more aggressive approach that may include ipsilateral vessel-sparing transpositions of the vertical rectus muscles or splitting transposition techniques of the vertical rectus muscles, with the goal of reduction or elimination of head turn. CN 3 palsies are among the most difficult to treat and should be referred to the most experienced strabismus surgeons.

Binenbaum et al. [99] examined the natural history of retinal hemorrhage (RH) in infants and young children with head trauma. In eyes with hemorrhage, intraretinal hemorrhage (IRH) was uniformly present on initial examination and was in many, but not all, cases accompanied by preretinal hemorrhage (PRH). IRHs resolved rapidly, often within 72 hours. Even in eyes with a large number of hemorrhages, including too numerous to count (TNTC), the vast majority resolve over a period of a few days. In his study, Binenbaum reported that by 2 weeks, either all IRHs had resolved or there were less than 10–20 hemorrhages even in eyes that presented with TNTC hemorrhages. PRHs resolved more slowly. The authors conclude that if PRHs are seen with no or very few IRHs, the injury is likely to have occurred at least a few days to a week or more prior to presentation. Conversely, if TNTC IRHs are observed on the initial examination, the injury very likely occurred within the past very few days, and as such, hemorrhages often resolved within a week. If 10–20 IRHs are present at the first examination, timing of the injury is more difficult and the insult may have occurred as recently as the past day or as long as 2 weeks earlier. These observations are consistent with studies of birth-related RHs [100, 101].

The longest a single IRH lasted was 32 days in the Binenbaum [99] study and 58 days in a report by Hughes et al. [100] In both of these cases, only a single large dense IRH persisted this long. Each author concluded that numerous or multiple grouped IRHs present in an infant past the age of 4 weeks are not consistent with birth trauma, and another etiology should be sought. Increasing severity of RH has been strongly associated with increasing specificity for abusive versus accidental traumatic injuries (Fig. 11.7), and the ability to obtain this diagnostic information may decrease in the RH pattern changes over time. The authors therefore recommend examinations be performed as soon as possible, preferably within 24–48 hours after the child presents to the hospital.

Shaken Baby (Impact) Syndrome

Caffey was the first to recognize an association of long bone fractures, subdural hemorrhages, and retinal hemorrhages in infants and coined the term whiplash shaken infant syndrome [102]. In cases of suspected child abuse (Fig. 11.8), the

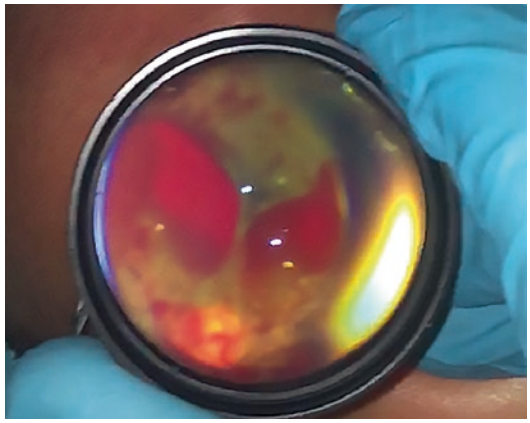

Fig. 11.7 Subretinal, intraretinal, and preretinal hemorrhages in a 4-month-old male infant

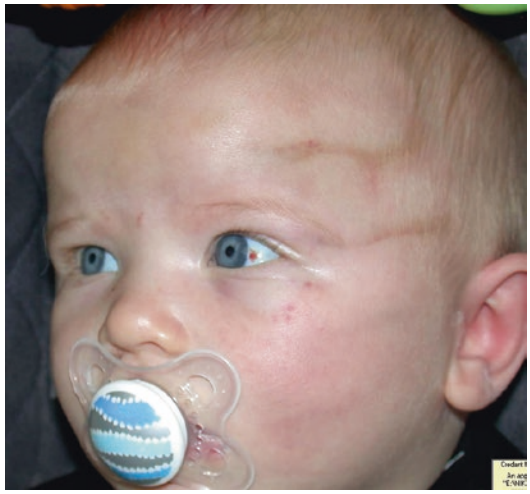

Fig. 11.8 Lower lid edema and ecchymosis, subconjunctival hemorrhage, and a distinct handprint caused by a slap in the face of this victim of NAT

role of the ophthalmologist is vital and may be the first provider to sound the alarm. Ocular manifestations may include lid edema and ecchymosis (Fig. 11.9), burns, abrasions, orbital fractures, corneal lacerations, hyphema, traumatic cataracts, multilayered (pre-, intra-, and subretinal) hemorrhages as well as optic nerve injury [103, 104]. This may also result in significant neurologic injury including cortical visual impairment, cranial nerve palsies, encephalopathy, and nystagmus [105].

Since Caffey's report back in the 1970s, we have learned that the incidence of retinal hemorrhage in victims of shaken baby (impact) syndrome (SBS) also referred to as nonaccidental trauma (NAT) is approximately 85% [102–104]. Although there is an association between severity of brain injury and retinal hemorrhage severity, retinal hemorrhages can rarely occur without intracranial hemorrhage or cerebral edema [102–104]. Approximately two-thirds of victims have RHs that are TNTC and multilayered [105]. Greenwald et al. [106] were the first to describe macular retinoschisis, where blood accumulates at the level of cleavage of the retinal internal limiting membrane and nerve fiber layer with occasional breakthrough into the vitreous cavity. Fortunately, retinal hemorrhage and schisis do not typically affect the patient's visual outcome. Although the incidence of retinal hemorrhage in SBS is dramatically higher than all forms of accidental blunt head injury, severe retinal hemorrhages have been reported in lethal motor vehicle accidents, especially roll-overs [81] with repeated acceleration–deceleration. Once thought to be pathognomonic for SBS, very rare reports of macular retinoschisis resulting from vitreous traction on the macula [107] with often coincident circumlinear traumatic retinal folds surrounding the schisis cavity can be found in the literature.

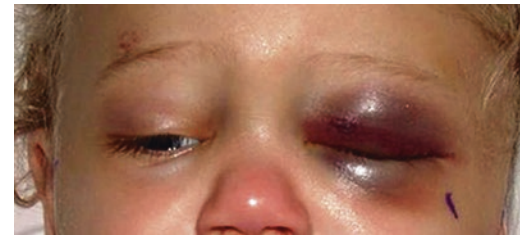

Fig. 11.9 Infant punched by a male adult

In a separate study, Binenbaum et al. [108] found an association between severity of retinal hemorrhage and hypoxic-ischemic brain injury (HII) patterns, as identified on diffusion weighted magnetic resonance imaging (DW-MRI), in young children with head trauma. Retinal hemorrhage was not correlated with the severity of traumatic intracranial injury even though evidence of traumatic injury was present in all cases with a predominantly post-traumatic HII pattern.

Moreover, HII was only seen in inflicted trauma. These findings suggest that a distinct type of traumatic injury may occur during inflicted head trauma that causes both greater global brain injury with HII and more severe retinal hemorrhage but not necessarily more severe intracranial injury. MRI is exquisitely sensitive in depicting the extent and anatomic distribution of hypoxic-ischemic parenchymal injury. HII in the setting of trauma may relate to poor systemic perfusion, decreased oxygenation, raised intracranial pressure, intravascular microthrombosis, reactive vasospasm adjacent to intracranial hemorrhage lesions, cervicomedullary injuries, strangulation, disrupted autoregulation, or apnea [109–112]. However, none of those factors in isolation has been shown to cause severe retinal hemorrhage. The pattern of HII should arouse one's suspicions of inflicted head trauma but is not a requisite factor for severe retinal hemorrhage, as severe retinal hemorrhages are noted in the absence of HII in children with inflicted head trauma [113].

Intrascleral hemorrhage can occur at the optic nerve–globe junction, another fulcrum point prone to tissue damage during repeated movement of the globe during SBS [114, 115]. The powerful association of severe hemorrhage with the unique repetitive acceleration/deceleration forces of SBS, as opposed to single-impact events, together with the findings of RH in areas of maximal vitreoretinal adhesion, strongly support causation by vitreoretinal traction. Retinal hemorrhages associated with intracranial hemorrhage after single-impact trauma are typically confined to the posterior pole [115]. A single acceleration/deceleration episode does not typically result in severe retinal hemorrhages, unless, in the very rare event, forces rise to the level of fatal impact or crush. However, in these cases, the history and cause of the hemorrhages are evident.

In the cases of suspected child abuse (Fig. 11.9), the role of the ophthalmologist is supportive. We examine the eyes and report our findings without assigning guilt. Ours is another, albeit significant, piece of the puzzle that is nonaccidental trauma. Retinal hemorrhage alone should rarely, if ever, be used to diagnose nonaccidental trauma without supportive historical, physical, radiographic, and laboratory evidence that could lead to the consideration of other etiologies. When in doubt, other conditions should be considered, such as increased intracranial pressure, hypoxia, sodium balance, bacterial meningitis, coagulopathy, anemia, birth trauma, leukemia, or other systemic disorders with associated retinal hemorrhage. If any of the aforementioned conditions are present, the history and physical findings are often so clear that, even with a skeptical mind, the diagnosis is (relatively) clear and the parents or caretakers should be given the benefit of the doubt.

Abuser's stories are often inconsistent and tend to change into what they think the investigators want to hear. Ophthalmologists who are presented with a previously healthy child who has become more irritable or somnolent in relatively short order, whose appetite is decreased, and/or whose parents express serious concern regarding the welfare of their infant, should heighten suspicions for NAT. Moreover, if your examination reveals any evidence of trauma, especially bilateral retinal hemorrhages with or without macular retinoschisis, your pediatric colleagues along with social services should be consulted immediately and an investigation launched to protect that child.

References

1. Abbott J, Shah P. The epidemiology and etiology of pediatric ocular trauma. Surv Ophthalmol. 2013;58(5):476–85.
2. Brophy M, Sinclair SA, Hostetler SG, Xiang H. Pediatric eye injury-related hospitalizations in the United States. Pediatrics. 2006;117(6):e1263–71.
3. Prevent Blindness America. Protect your child's eyes. Available at: https://www.preventblindness.org/protect-your-child-eye-injuries. Accessed 17 Sep 2017.
4. Prevent Blindness America. Leading cause of eye injuries in school-aged children are sports-related. Available at: https://www.preventblindness.org/leading-cause-eye-injuries-school-aged-children-are-sports-related. Accessed 17 Sep 2017.
5. Moren Cross J, Griffin R, Owsley C, McGwin G Jr. Pediatric eye injuries related to consumer prod-

ucts in the United States, 1997-2006. J AAPOS. 2008;12(6):626–8.
6. Saunte JP, Saunte ME. Childhood ocular trauma in the Copenhagen area from 1998to 2003: eye injuries caused by airsoft guns are twice as common as firework-related injuries. Acta Ophthalmol. 2008;86(3):345–7.
7. Garcia TA, McGetrick BA, Janik JS. Spectrum of ocular injuries in children with major trauma. J Trauma. 2005;59(1):169–74.
8. Oiticica-Barbosa MM, Kasahara N. Eye trauma in children and adolescents: perspectives from a developing country and validation of the ocular trauma score. J Trop Pediatr. 2015;61(4):238–43.
9. Haavisto AK, Sahraravand A, Holopainen JM, Leivo T. Paediatric eye injuries in Finland – Helsinki eye trauma study. Acta Ophthalmol. 2017;95(4):392–9.
10. Scott R. The ocular trauma score. Community Eye Health. 2015;28(91):44–5.
11. Salvin JH. Systematic approach to pediatric ocular trauma. Curr Opin Ophthalmol. 2007;18(5):366–72.
12. Harlan JB Jr, Pieramici DJ. Evaluation of patients with ocular trauma. Ophthalmol Clin N Am. 2002;15(2):153–61.
13. Root J, Gupta S, Jamal N. Non-penetrating eye injuries in children. Clin Pediatr Emerg Med. 2017;18(1):74–86.
14. McGwin G Jr, Owsley C. Incidence of emergency department-treated eye injury in the United States. Arch Ophthalmol. 2005;123(5):662–6. Review.
15. Lee R, Fredrick D. Pediatric eye injuries due to nonpowder guns in the United States, 2002–2012. J AAPOS. 2015;19(2):163–8.e1.
16. Napier SM, Baker RS, Sanford DG, Easeterbook M. Eye injuries in atheletics and recreation. Surv Ophthalmol. 1996;41(3):229–44.
17. Agapitos PJ, Noel LP, Clarke WN. Traumatic hyphema in children. Ophthalmology. 1987;94:1238–41.
18. Hoskin AK, Philip SS, Yardley AM, Mackey DA. Eye injury prevention for the pediatric population. Asia Pac J Ophthalmol (Phila). 2016;5(3):202–11.
19. Mowatt L. Epidemiology of pediatric ocular trauma admissions. Surv Ophthalmol. 2014;59(4):480.
20. Billock RM, Chounthirath T, Smith GA. Pediatric firework-related injuries presenting tot the United States Emergency Departments, 1990-2014. Clin Pediatr (Phila). 2017;56(6):535–44.
21. Khan M, Reichstein D, Recchia FM. Ocular consequences of bottle rocket injuries in children and adolescents. Arch Ophthalmol. 2011;129(5):639–42.
22. Ramstead C, Ng M, Rudnisky CJ. Ocular injuries associated with Airsoft guns: a case series. Can J Ophthalmol. 2008;43(5):584–7.
23. Listman DA. Paintball injuries in children: more than meets the eye. Pediatrics. 2004;113(1 Pt 1):e15–8.
24. Vajpayee RB, Shekhar H, Sharma N, Jhanji V. Demographic and clinical profile of ocular chemical injuries in the pediatric age group. Ophthalmology. 2014;121(1):377–80.
25. Yardley AM, et al. Animal-inflicted ocular and adnexal injuries in children: a systematic review. Surv Ophthal mol. 2015;60(6):536–46.
26. Berríos RR, Serrano LA. Bilateral optic neuritis after a bee sting. Am J Ophthalmol. 1994;117(5):677–8.
27. Maltzman JS, Lee AG, Miller NR. Optic neuropathy occurring after bee and wasp sting. Ophthalmology. 2000;107(1):193–5.
28. Shriwas SR. Bird-related eye injuries. Trop Dr. 1993;23(3):140.
29. Tabatabaei SA, Soleimani M, Behrouz MJ. Bird attack ocular injuries. Retina. 2018;38(5):945–50.
30. O'Neill JF. Eye safety in the pediatric population. Ophthalmol Clin. 1999;12(3):413–9.
31. Coon D, Yuan N, Jones D, Howell LK, Grant MP, Redett RJ. Defining pediatric orbital roof fractures: patterns, sequelae, and indications for operation. Plast Reconstr Surg. 2014;134(3):442e–8e. https://doi.org/10.1097/PRS.0000000000000421.
32. Hink EM, Wei LA, Durairaj VD. Clinical features and treatment of pediatric orbit fractures. Ophthal Plast Reconstr Surg. 2014;30(2):124–31. https://doi.org/10.1097/IOP.0000000000000026.
33. Phan LT, Jordan Piluek W, McCulley TJ. Orbital trapdoor fractures. Saudi J Ophthalmol. 2012;26(3):277–82. https://doi.org/10.1016/j.sjopt.2012.05.008. Epub 2012 Jun 13.
34. Coon D, Kosztowski M, Mahoney NR, Mundinger GS, Grant MP, Redett RJ. Principles for management of orbital fractures in the pediatric population: a cohort study of 150 patients. Plast Reconstr Surg. 2016;137(4):1234–40. https://doi.org/10.1097/PRS.0000000000002006.
35. Mehta VJ, Chelnis JG, Chen Q, Mawn LA. Effect of time to operative intervention on motility outcomes following orbital floor fracture repair in children. Ophthal Plast Reconstr Surg. 2018;34(4):351–4. https://doi.org/10.1097/IOP.0000000000000993.
36. Prendes MA, Jian-Amadi A, Chang SH, Shaftel SS. Ocular trauma from dog bites: characterization, associations, and treatment patterns at a regional level I trauma center over 11 years. Ophthal Plast Reconstr Surg. 2016;32(4):279–83. https://doi.org/10.1097/IOP.0000000000000501.
37. Bratton EM, Golas L, Wei LA, Davies BW, Durairaj VD. Ophthalmic manifestations of facial dog bites in children. Ophthal Plast Reconstr Surg. 2018;34(2):106–9. https://doi.org/10.1097/IOP.0000000000000875.
38. Michael JG, Hug D, Dowd MD. Management of corneal abrasion in children: a randomized clinical trial. Ann Emerg Med. 2002;40(1):67–72.
39. Mittal V, Jain R, Mittal R, Vashist U, Narang P. Successful management of severe unilateral chemical burns in children using simple limbal epithelial transplantation (SLET). Br J Ophthalmol. 2016;100(8):1102–8. https://doi.org/10.1136/bjophthalmol-2015-307179. Epub 2015 Dec 23.
40. Karolinne MR, Nogueira Martins E, Melo LAS Jr, Bueno de Moraes NS. Outpatient management of

traumatic hyphema in children: prospective evaluation. J AAPOS. 2004;8:357–61.
41. Walton W, Von Hagen SV, Grigorian R, Zarbin M. Management of traumatic hyphema. Surv Ophthalmol. 2002;47:297–334.
42. Berrios RR, Dreyer EB. Traumatic hyphema. Int Ophthalmol Clin. 1995;35:93–103.
43. Crouch ER Jr, Crouch ER. Management of traumatic hyphema: therapeutic options. J Pediatr Ophthalmol Strabismus. 1999;102:1646–53.
44. Edwards WC, Layden WE. Traumatic hyphema. Am J Ophthalmol. 1973;75:110–6.
45. Agaptios PJ, Leon-Paul N, Clarke WN. Traumatic hyphema in children. Ophthalmology. 1987;94:1238–41.
46. Reddy AK, Ray R, Yen KG. Surgical intervention for traumatic cataracts in children: epidemiology, complications and outcomes. J AAPOS. 2009;13:170–4.
47. Hargrave S, Weakley D, Wilson C. Complications of ocular paintball injuries in children. J Pediatr Ophthalmol Strabismus. 2000;37:338–43.
48. Thach AB, Ward TP, Hollifield RD, et al. Ocular injuries from paintball pellets. Ophthalmology. 1999;106:533–7.
49. Zaidman GW, Ramirez TC, Kaufman AH, et al. Successful surgical rehabilitation of children with traumatic corneal laceration and cataract. Ophthalmology. 2001;108:338–42.
50. Sindal MD, Nakhwa CP, Sengupta S. Comparison of sutured versus sutureless scleral-fixated intraocular lenses. J Cataract Refract Surg. 2016;42:27–34.
51. Evereklioglu C, Er H, Bekir NA, Borazan M, Zorlu F. Comparison of secondary implantation of flexible open-loop anterior chamber and scleral-fixated posterior chamber intraocular lenses. J Cataract Refract Surg. 2003;26:301–8.
52. Wagoner MD, Cox TA, Ariyasy RG, Jacobs DS, Karp CL. Intraocular lens implantation in the absence of capsular support; a report by the American Academy of Ophthalmology (Ophthalmic Technology Assessment). Ophthalmology. 2003;110:840–59.
53. Chan TCY, Jam JKM, Jahnji V, Li EYM. Comparison of outcomes of primary anterior chamber versus secondary scleral-fixated intraocular lens implantation in complicated cataract surgeries. Ophthalmology. 2015;159:221–6.
54. Price MO, Price FW Jr, Werner L, Berlie C, Mamalis N. Late dislocation of scleral-sutured posterior chamber intraocular lenses. J Cataract Refract Surg. 2005;31:1320–6.
55. Stewart M, Landers M. Transscleral intraocular lens fixation with a “homemade” needle and hook. J Cataract Refract Surg. 2006;32:200–2.
56. Buckley EG. Safety of transscleral sutured intraocular lenses in children. J AAPOS. 2008;12:431–9.
57. Gabor SGB, Pavlidis MM. Sutureless intrascleral posterior chamber intraocular lens fixation. J Cataract Refract Surg. 2007;33:1851–4.
58. Abbey AM, Hussain RM, Shah AR, Faia LJ, Wolfe JD, Williams GA. Sutureless scleral fixation of intraocular lenses: outcomes of two approaches. The 2014 Yasuo Tano Memorial Lecture. Graefes Arch Clin Exp Ophthalmol. 2014;253:1–5.
59. Snyder ME, Perez MA. Tiltless and centration adjustable scleral-sutured posterior chamber intraocular lens. J Cataract Refract Surg. 2014;40:1579–83.
60. Greenwald MJ, Glaser SR. Visual outcomes after surgery for unilateral cataract in children more than two years old: posterior chamber intraocular lens implantation versus contact lens correction of aphakia. J AAPOS. 1998;2:168–76.
61. Dana MR, Werner MS, Viana MA, Shapiro MJ. Spontaneous and traumatic vitreous hemorrhage. Ophthalmology. 1993;100:1377–83.
62. Spirn MJ, Lynn MJ, Hubbard GB III. Vitreous hemorrhage in children. Ophthalmology. 2006;113:848–52.
63. Sebag P. Anomalous posterior vitreous detachment: a unifying concept in vitreo-retinal disease. Graefes Arch Clin Exp Ophthalmol. 2004;242:690–8.
64. Manschot WA. Retinal detachment in young children. Ophthalmologica. 1961;141:47–50.
65. Hudson JR. Retinal detachments in children. Trans Ophthalmol Soc U K. 1965l;85:79–91.
66. Tasman W. Retinal detachment in children. Trans Am Acad Ophthalmol Otolaryngol. 1967;71:455–60.
67. Okinami S, Ogino N, Nishimura T, et al. Juvenile retinal detachment. Ophthalmologica. 1987;194:95–102.
68. AL-Zaaidi S, AL-Rashaed S, AL-Hathi E, AL-Kahtani E, Abu El-Asrar AM. Rhegmatogenous retinal detachment in children 16 years of age or younger. Clin Ophthalmol. 2013;7:1001–14.
69. Sarrazin L, Averbukh E, et al. Traumatic pediatric retinal detachment: a comparison between open and closed globe injuries. Am J Ophthalmol. 2004;137:1042–9.
70. Winslow RL, Tasman W. Juvenile rhegmatogenous retinal detachment. Ophthalmology. 1978;85:607–18.
71. Brinton GS, Aaberg TM, Reeser FH, et al. Surgical results in ocular trauma involving the posterior segment. Am J Ophthalmol. 1982;93:271–8.
72. Greven CM, Tasman W. Rhegmatogenous retinal detachment following cryotherapy in retinopathy of prematurity. Arch Ophthalmol. 1989;107:1017–8.
73. Rosner M, Treister G, Belkin M. Epidemiology of retinal detachment in childhood and adolescence. J Pediatr Ophthalmol Strabismus. 1987;24:42–4.
74. Akabane N, Yamamoto S, Tsukahara I, et al. Surgical outcomes in juvenile retinal detachment. Jpn J Ophthalmol. 2001;45:409–11.
75. Kiffney GT Jr. The eye of the “battered child”. Arch Ophthalmol. 1964;72:231–3.
76. Fivgas GD, Capone A Jr. Paediatric rhegmatogenous retinal detachment. Retina. 2001;21:101–6.
77. Go SL, Hoyng CB, Klaver CC. Genetic risk of rhegmatogenous retinal detachment: a familial aggregation study. Arch Ophthalmol. 2005;123:1237–41.
78. Pieramici DJ, Sternberg P Jr, Aaberg TM Sr, Bridges WZ Jr, Capone A Jr, Cardillo JA, et al. The ocular trauma classification group. A system for classify-

ing mechanical injuries of the eye (globe). Am J Ophthalmol. 1997;123:820–31.
79. Farr AK, Hairston RJ, Humayun MU, et al. Open globe injuries in children: a retrospective analysis. J Pediatr Ophthalmol Strabismus. 2001;38:72–7.
80. Rudd JC, Jaeger EA, Freitag SK, Jeffers JB. Traumatically ruptured globes in children. J Pediatr Ophthalmol Strabismus. 1994;31:307–11.
81. Scott IU, Flynn HW, Azen SP, et al. Silicone oil in the repair of pediatric complex retinal detachments. A prospective, observational, multicenter study. Ophthalmology. 1999;106:1399–408.
82. Segev FS, Assia EI, Harizman N, et al. Corneal laceration by sharp objects in children seven years of age and younger. Cornea. 2007;26:319–23.
83. Cascairo MA, Mazow ML, Prager TC. Pediatric ocular trauma: a retrospective survey. J Pediatr Ophthalmol Strabismus. 1994;31:312–7.
84. De Juan E Jr, Steinberg P Jr, Michels RG. Penetrating ocular injuries: types of injuries and visual results. Ophthalmology. 1983;90:1318–22.
85. Barr CC. Prognostic factors in corneoscleral lacerations. Arch Ophthalmol. 1983;101:919–24.
86. Li X, Zarbin MA, Bhagat N. Pediatric open globe injury: a review of the literature. J Emerg Trauma Shock. 2015;8(4):216–23.
87. May DR, Kuhn FP, Morris RE, Witherspoon CD, et al. The epidemiology of serious eye injuries from the United States eye injury registry. Graefes Arch Clin Exp Ophthalmol. 2000;238:153–7.
88. Lee CH, Su WY, Lee L, Yang ML. Pediatric ocular trauma in Taiwan. Chang Gung Med J. 2008;31:59–65.
89. Sheard RM, Mireskandari K, Ezra E, Sullivan PM. Vitreoretinal surgery after childhood ocular trauma. Eye (Lond). 2007;21:793–8.
90. Hosseini H, Masoumpour M, Keshavarz-Fazl F, Razeghinejad MR, Salouti R, Nowroozzadeh MH. Clinical and epidemiologic characteristics of severe childhood ocular injuries in southern Iran. Middle East Afr J Ophthalmol. 2001;19:186–91.
91. Kumar K, Mathai A, Murthy SI, Jalali S, et al. Sympathetic ophthalmia in pediatric age group: clinical features and challenges in management in a tertiary center in southern India. Ocul Immunol Inflamm. 2014;22:367–72.
92. Raina UK, Kumar V, Sud R, Goel N, Ghosh B. Metallic intraocular foreign body retained for four years – an unusual presentation. Cont Lens Anterior Eye. 2010;33:202–4.
93. Parke DW 3rd, Flynn HW Jr, Fisher YL. Management of intraocular foreign bodies: a clinical flight plan. Can J Ophthalmol. 2013;48:8–12.
94. Sangermani C, Mora P, Mancini C, Vecchi M, Gandolfi SA. Ultrasound biomicroscopy in two cases of ocular sidersosis with secondary glaucoma. Acta Opthalmol. 2010;88:e-2.
95. Ballantyne JF. Sidersosis bulbi. Br J Ophthalmol. 1954;38:727–33.
96. Lim LT, Shankar V, Blum RA, Hammer HM. Long-standing iron-containing intraocular foreign body without siderosis. Clin Exp Optom. 2011;94:387–8.
97. Bhagat N, Nagor S, Zarbin M. Post-traumatic infectious endophthalmitis. Surv Ophthalmol. 2011;56:214–51.
98. Polland KA, Xiang H, Smith GA. Pediatric eye injuries treated in US emergency departments, 1990–2009. Clin Pediatr (Phila). 2012;51:374–81.
99. Binenbaum G, Chen W, et al. The natural history of retinal hemorrhage in pediatric head trauma. J AAPOS. 2016;20:131–5.
100. Watts P, Maguire S, Kwok T, et al. Newborn retinal hemorrhages: a systematic review. J AAPOS. 2013;17:70–8.
101. Caffey J. The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash induced intracranial and intraocular bleedings linked with residual permanent brain damage and mental retardation. Pediatrics. 1974;54:396–403.
102. Levin AV. Retinal hemorrhage in abusive head trauma. Pediatrics. 2010;126:961–70.
103. Kivlin J, Simons K, Lazoritz S, Ruttum M. Shaken baby syndrome. Ophthalmology. 2000;107(7):1246–54.
104. Morad Y, Kim Y, Armstrong D, Huyer D, Mian M, Levin A. Correlation between retinal abnormalities and intracracnial abnormalities in the shaken baby syndrome. Am J Ophthalmol. 2002;134(3):354–9.
105. Levin A. Retinal haemorrhage and child abuse. In: Davie T, editor. Recent advances in paediatrics, vol. 18. London: Churchill Livingstone; 2000. p. 151–219.
106. Greenwald M, Weiss A, Oesterle C, Friendly D. Traumatic retinoschisis in battered babies. Ophthalmology. 1986;93(5):618–25.
107. Kivlin JD, Currie ML, Geenbaum VJ, Simons KB, Jentzen J. Retinal hemorrhages in children following fatal motor vehicle crashes: a case series. Arch Ophthalmol. 2008l;126(6):800–80.
108. Binenbaum G, Christian CW, Ichord RN, et al. Retinal hemorrhage and brain injury patterns on diffusion-weighted magnetic resonance imaging in children with head trauma. J AAPOS. 2013;17:603–8.
109. Biousse V, Suh DY, Newman NJ, Davis PC, Mapstone T, Lambert SR. Diffusion-weighted magnetic resonance imaging in shaken baby syndrome. Am J Ophthalmol. 2002;133:249–55.
110. Ichord RN, Naim M, Pollock AN, Nance NL, Margulies SS, Christian CW. Hypoxic-ischemic injury complicates inflicted and accidental traumatic brain injury in young children: the role of diffusion-weighted imaging. J Neurotrauma. 2007;24:16–118.
111. Kemp AM, Stoodley N, Cobley C, Coles L, Kemp KW. Apnoea and brain swelling in non-accidental head injury. Arch Dis Child. 2003;88:472–6; discussion 472–6.
112. Parizel PM, Ceulemans B, Laridon A, Ozsarlak O, Van Goethem JW, Jorens PG. Cortical hypoxic-

ischemic brain damage in shaken-baby (shaken impact) syndrome: value of diffusion-weighted MRI. Pediatr Radiol. 2003;33:868–71.
113. Morad Y, Avni I, Benton S, et al. Normal computerized tomography of brain in children with shaken baby syndrome. J AAPOS. 2004;8(5):445–50.
114. Morad Y, Avni I, Capra L, et al. Shaken baby syndrome without intracranial hemorrhage on initial computed tomography. J AAPOS. 2004;8(6):521–7.
115. Lin K, Glasgow B. Bilateral periopticintrascleral hemorrhages associated with traumatic child abuse. Am J Ophthalmol. 1999;127(4):473–5.

Uveitis

12

R. Christopher Walton

Abbreviations

CT Computed tomography
LIU Lens-induced uveitis

Traumatic Uveitis

Traumatic uveitis typically occurs following blunt trauma to the globe. Previously termed traumatic iritis or traumatic iridocyclitis, it can be a transient, self-limited event, or a prolonged inflammatory response resistant to typical anti-inflammatory therapy [1]. Traumatic uveitis is also frequently seen in association with other injuries following penetrating ocular trauma. This is a common presentation in patients following war injuries, terror attacks, and natural disasters. In these situations, the uveitis is often obscured by other traumatic injuries such as hyphema, iris trauma, or iris incarceration into a wound.

Isolated traumatic uveitis is less common in combat and civilian casualty situations compared to other ocular blunt injuries. In many cases, the object is larger than the globe and tends to be a lower velocity impact compared to penetrating wounds [2]. However, smaller, higher velocity objects such as BB and plastic pellets may produce blunt trauma and traumatic uveitis [3]. Hands, feet, other body parts, balls, paintballs, rackets, and various other blunt objects can strike the globe during assaults, home-related or sporting activities, or work-related activities [4–7].

Traumatic uveitis may also occur as a result of primary blast injury to the eye. Primary blast injury is the result of a strong overpressurization following the blast or shock wave that leads to deformation, disruption, and possible rupture of tissues. Less powerful overpressurization may cause disruption of internal ocular structures [8]. Secondary blast injuries (flying debris and/or fragments) produce most of the ocular injuries following explosions and bombings [8, 9]. Review of the literature does not reveal any reports of traumatic uveitis as an isolated injury following a primary blast injury. However, traumatic uveitis may be one of several closed-globe injuries that occur following explosive blasts and may be underreported or undetected due to presence of these other injuries such as hyphema [10–15].

Symptoms of isolated traumatic uveitis include blurry vision, photophobia, redness, and occasionally pain. Some patients may have an associated corneal abrasion. Traumatic corneal endothelial rings may be visible [16–18]. All patients exhibit anterior chamber cells and flare. Pigmented cells are often seen in the anterior

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chamber and a microhyphema may be present. In severe cases, fibrin and posterior synechiae may be present. In some patients, small tears of the iris along the pupil margin are visible, as well as other signs of iris trauma [4]. Intraocular pressure may be increased or decreased. Rarely, glaucoma may develop as a result of prolonged inflammation or therapy with corticosteroids.

Initial treatment of isolated traumatic uveitis is based upon the patient's level of discomfort and the severity of the inflammation. In most patients, a topical cycloplegic will reduce ciliary spasm and relieve pain and photophobia. In more severe cases, a cycloplegic agent is often useful for prevention of posterior synechiae formation. Topical corticosteroids are the mainstay of therapy in most patients with mild to severe forms of traumatic uveitis. In cases with severe inflammation with or without fibrin, hourly prednisolone acetate 1% is recommended. Periocular triamcinolone acetonide injections may be beneficial for those patients with severe inflammation unresponsive to topical corticosteroids. Topical corticosteroid dosage is reduced and tapered off beginning after a 50% reduction in the amount of cell and flare is noted. Patients with severe inflammation may require prolonged treatment for several months with topical corticosteroids before tapering, and discontinuation may occur. Topical glaucoma therapy may be required if the intraocular pressure is elevated. Topical beta-blockers, carbonic anhydrase inhibitors, and alpha-agonists are useful options for patients who are at risk for vision loss due to elevated intraocular pressure [19, 20].

In patients with traumatic uveitis and other anterior segment traumatic injuries, the diagnosis and management of the uveitis is more complex. Most of these cases occur in the setting of penetrating trauma with coexisting pathology such as corneal laceration, iris incarceration and/or prolapse, hyphema, iris trauma, lens capsule disruption, lens dislocation, and other anterior segment abnormalities. These injuries often limit evaluation of the anterior chamber and any underlying uveitis. Therefore, careful evaluation of the anterior chamber following repair of any corneal wounds or removal of lens material is essential to determine the severity of the uveitis and initial therapeutic approach. Similar to isolated traumatic uveitis, topical prednisolone acetate 1% is prescribed based upon the severity of the inflammation. A topical cyloplegic agent may also be considered based upon the clinical situation. In most cases of penetrating trauma requiring surgical repair, a slow taper of corticosteroids over several months may be necessary based upon the severity of uveitis during follow-up evaluations. Elevated intraocular pressure may require treatment with topical glaucoma medications, as discussed previously.

Complications of mild isolated traumatic uveitis without additional intraocular damage are uncommon. Some patients with isolated traumatic uveitis may have a severe prolonged inflammatory response requiring months of topical corticosteroid therapy and slow taper. These patients may be at increased risk for development of cataract as well as secondary glaucoma. Patients with severe blunt trauma often have concomitant injuries such as hyphema, angle recession, inflammatory trabeculitis, or other conditions that may lead to complications. In these patients, synechiae formation, secondary glaucoma, and traumatic cataract may occur as a result of these additional injuries.

Patients with traumatic uveitis and globe trauma are at increased risk for complications, typically due to the perforating/penetrating injuries and subsequent inflammatory response. Complications resulting from the globe trauma may result in synechiae formation, secondary glaucoma, cataract, as well as cyclitic membrane formation. Some patients may experience unremitting anterior uveitis following successful repair of the corneal and/or scleral wounds. In these cases, an occult foreign body should be suspected. Ultrasound is often useful to locate the foreign body, although in some patients computed tomography (CT) may be necessary [21–23]. Magnetic resonance imaging may also be useful for detection of occult non-metallic foreign bodies if initial CT scanning is negative [24]. Persistent inflammation may also occur in patients with chronic endophthalmitis following trauma. This is an atypical presenta-

tion of post-traumatic endophthalmitis, and the diagnosis is often delayed. In these patients, samples of aqueous and vitreous are typically obtained for culture and sensitivities. The etiology in these unusual cases may include bacteria as well as fungi. Treatment is guided by culture and sensitivity results (see section "Traumatic Endophthalmitis").

Lens-Induced Uveitis

Lens-induced uveitis (LIU) includes several types of uveitis that differ in etiology and pathogenesis. In this chapter, the term LIU will be used to describe any inflammatory process resulting from trauma to the lens. This includes blunt trauma as well as perforating and penetrating injuries to the lens. Older terms for these types of uveitis include phacoanaphylaxis, phacoanaphylactic endophthalmitis, phacoantigenic, and phacogenic uveitis.

The pathogenesis of LIU is uncertain; however, older theories are primarily based upon an autoimmune response to released lens proteins [25–28]. The proposed triggering mechanism in virtually all cases is traumatic rupture of the lens capsule [29]. Histopathology reveals an area of zonal granulomatous inflammation surrounding the lens material and ruptured lens capsule. Epithelioid and giant cells are also present in the zone of inflammation with a surrounding mononuclear cell infiltrate. The uvea is also infiltrated with lymphocytes as well as plasma cells [25, 29, 30].

Blunt trauma to the anterior segment can produce rapid posterior movement of the lens-iris diaphragm without apparent damage to the lens. However, in some cases, the trauma may cause rupture of the lens capsule with release of lens proteins into the anterior chamber [16, 31, 32]. Anterior chamber cells and flare are visible and a hypopyon may be present. Moderate to severe anterior uveitis is typical and lens material may be seen in the anterior chamber in some cases (Fig. 12.1). In most patients, fine or mutton-fat keratic precipitates are noted. A tear in the anterior lens capsule may be visible, although most cases will have a wrinkled anterior lens capsule surface. Additional injuries such as corneal abrasions or hyphema are common and may obscure assessment of the anterior segment structures and the lens-induced inflammatory response.

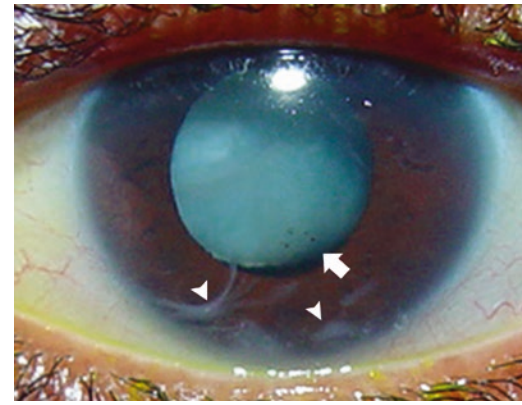

Fig. 12.1 Lens-induced uveitis following blunt trauma to the eye. Lens material (arrowheads) is visible in the inferior anterior chamber. Superior dislocation of lens (arrow) is also noted

Perforating or penetrating injuries to the lens may also damage the lens with liberation of lens protein into the anterior chamber as well as release of cortical and/or nuclear lens material [33]. The resultant inflammatory response may be mild to severe depending upon the amount of material released [34]. In many cases, multiple anterior segment structures are damaged at the time of injury. As a result, a mixed inflammatory reaction is often present in the anterior chamber with inflammatory cells and blood as well as tissue fragments and possibly foreign bodies. With most of these severe injuries, lens material may not be visible initially and only later discovered as the inflammation and blood clears. In some cases, the disorder is unrecognized and phthisis may eventually result [16, 25, 29, 30].

Treatment of LIU whether from blunt or penetrating injury is surgical removal of all lens material [25, 34, 35]. The timing of surgical removal is based upon several factors, including the presence of visible lens material in the anterior chamber, extent of lens damage, concomitant anterior segment injuries, visibility of the anterior chamber, as well as availability of equip-

ment for lens removal. In cases with extensive lens damage and large amounts of lens material in the anterior chamber, surgical removal should be performed at the same time as other injuries such as corneal laceration are repaired [29, 36, 37]. Removal of lens material may be delayed in patients without other injuries requiring urgent surgical repair [38]. Patients with small corneal lacerations and minimal lens trauma or cases with severe corneal edema or large hyphemas are examples of situations in which delayed surgical removal of lens material may be considered [37]. However, surgery should be performed as soon as possible in most patients to prevent worsening of lens-induced inflammation with time. Topical corticosteroids should be utilized to control the inflammation prior to and following surgery. Systemic corticosteroids may be necessary for some patients with severe inflammation inadequately controlled with topical therapy [25, 29].

Complications are relatively common, especially if there is a delay in diagnosis and treatment of traumatic LIU. Corneal edema may develop as a result of retained lens material in the anterior chamber or if there is an increase in intraocular pressure. Posterior synechiae as well as pupillary membrane formation are frequent in cases with uncontrolled inflammation. Some patients may develop secondary glaucoma due to the uveitis or chronic use of corticosteroids. In severe cases, a cyclitic membrane may develop leading to hypotony and ultimately phthisis [39, 40].

Traumatic Endophthalmitis

Infectious endophthalmitis is a rare but potentially devastating complication of penetrating and perforating injuries to the globe. Virtually all of these cases are exogenous endophthalmitis following trauma to the globe, although in combat and civilian disasters, multiple systemic injuries are common which may result in systemic infections leading to endogenous endophthalmitis [41–48]. In this chapter, traumatic endophthalmitis will refer to only those cases which are exogenous in nature, resulting from penetrating or perforating wounds.

The incidence of post-traumatic endophthalmitis has been estimated to be 0.9–17% [49–59]. Following combat-related injuries, the estimated incidence is 2–7.5% [12, 33, 60–64]. During the most recent US Military conflicts in Afghanistan and Iraq, no cases of traumatic endophthalmitis have been reported [64]. The incidence of endophthalmitis following open-globe injuries varies by several factors, including location (urban, rural, combat zone, etc.), presence of retained intraocular foreign body, lens capsule rupture, and delay in wound closure [50, 52, 56, 65]. In addition, estimated incidence rates may be less accurate in reports describing civilian or military mass casualty events due to the initial chaos of the event, limited time for meticulous data collection, and sparsity of follow-up data [66–71]. Rapid evacuation of casualties to higher levels of care at distant locations also contributes to the limited access to follow-up data [12, 64, 69].

The prognosis in cases of post-traumatic endophthalmitis varies by the virulence of the causative organism, extent of ocular injury, timing of treatment, presence of an intraocular foreign body, and presence of a retinal detachment [72–75]. Patients with poor initial visual acuity may also have a worse prognosis [75, 76]. Delays in primary repair greater than 24–72 hours are associated with worse visual outcomes [74, 77]. Compared with earlier studies, recent reports suggest that delayed removal of intraocular foreign bodies may not be associated with a worse prognosis in some situations [53, 63, 73, 78–80]. Overall, post-traumatic fungal endophthalmitis has a worse prognosis than bacterial endophthalmitis with over two-thirds of eyes having no light perception and many ultimately requiring evisceration or enucleation [81, 82].

Clinical manifestations of traumatic endophthalmitis are highly variable and may be masked by the underlying injury. As a result, delays in diagnosis are not uncommon. Patients typically complain of increasing pain with decreased vision during the first few days following injury or repair of the ruptured globe. However, in patients with fungal endophthalmitis, the onset of pain may be delayed for weeks to months following the injury. Common signs of endophthalmitis

include conjunctival hyperemia, severe anterior chamber cells and flare, corneal edema, and vitritis out of proportion to the underlying injury. Additional findings include purulent discharge from the site of injury, anterior chamber fibrin, hypopyon, infiltrates in the anterior chamber, or vitreous and retinal perivasculitis [74, 83–85]. In severe cases of post-traumatic endophthalmitis, panophthalmitis may develop if the infection spreads to the adjacent orbital structures. In these patients, eyelid edema and erythema, proptosis, pain with eye movement, and limited ocular motility are common [74]. Patients with *Bacillus cereus* endophthalmitis may have several unique clinical features, including severe eyelid edema, proptosis, corneal ring infiltrate or abscess, and in some cases fever and leukocytosis. These eyes may also demonstrate rapid progression of the infection with devastating visual loss. *Clostridium perfringens* is a much less common cause of endophthalmitis following trauma, but it too may have unusual clinical manifestations. Severe ocular pain, a green-brown hypopyon, and possibly intraocular gas bubbles are characteristic of infection with *Clostridium perfringens* [74, 86]. Rapid progression to panophthalmitis is not uncommon in these patients.

If traumatic endophthalmitis is suspected, specimens should be quickly obtained for stains, cultures, and antimicrobial sensitivities. If possible, specimens should be obtained prior to beginning antimicrobial therapy. However, in combat or civilian disasters, many patients have multiple systemic injuries and likely have received prophylactic antibiotics prior to the suspicion of endophthalmitis. Samples from the ocular wound, anterior chamber, and vitreous should be immediately inoculated onto appropriate microbiologic media. Vitreous specimens may be obtained by vitreous tap or biopsy [87]. Blood agar is used for detection of aerobic bacteria and fungi, while chocolate agar is utilized for aerobic bacteria as well as *Moraxella* and *Haemophilus* species. Thioglycolate broth is useful for anaerobic bacteria as well as microaerophilic bacteria. Sabouraud's dextrose agar is typically used for isolation of fungi [88–93]. Microscopic examination of Gram's stained specimens may reveal inflammatory cells as well as bacteria and their morphology and staining patterns. Staining with calcofluor-KOH should be performed in cases suspicious for fungal endophthalmitis [93, 94].

The causative organisms in most cases of traumatic endophthalmitis are bacteria. However, the organisms vary by location, contamination of the wound, and presence or absence of intraocular foreign bodies. Furthermore, infection with multiple organisms is not uncommon following traumatic injuries [50–52, 92, 95, 96]. Overall, gram-positive organisms are the most common pathogens in post-traumatic endophthalmitis accounting for approximately 75% of all cases [74, 97]. In many studies, *Staphylococcal epidermidis* is the most common gram-positive organism [52, 72, 74, 98–101]. This is not unexpected since *S. epidermis* is a normal constituent of the skin flora. Less common gram-positive bacteria include *S. aureus*, Streptococcal species, *Bacillus cereus*, other *Bacillus* species, and *Clostridium* species [51, 72, 74, 98, 101–106]. *Bacillus* species have been reported to account for up to 20% of post-traumatic endophthalmitis cases [74, 97, 102, 104, 105, 107]. *Bacillus cereus* is especially virulent with a rapid onset, usually within 24 hours following injury and often with devastating outcomes [108–111]. *Clostridium* species are gram-positive anaerobic bacteria which have the ability to form spores. *Clostridium perfringens* is found in soil throughout the world and is a known cause of post-traumatic endophthalmitis. Most cases of *Clostridium perfringens* endophthalmitis are associated with retained intraocular foreign bodies and have a very poor prognosis [74]. Gram-negative organisms are a less common cause of endophthalmitis accounting for up to 33% of cases of post-traumatic endophthalmitis [74, 98, 109]. Gram-negative organisms associated with endophthalmitis following trauma include *Pseudomonas* species, *Proteus mirabilis*, *Stenotrophomonas maltophilia*, *Acinetobacter*, and *Moraxella* [50, 72, 112–114]. Fungi are rare causes of post-traumatic endophthalmitis occurring in up to 15% of cases [72, 91]. Wounds contaminated by vegetable matter, mud, or stones may be at increased risk for fungal endophthalmitis [85, 115]. *Candida* species are the most

common fungal organisms isolated following globe trauma [90]. Specific fungal etiologies reported following globe trauma include *Candida albicans*, *Candida parapsilosis*, *Aspergillus fumigatus*, *Aspergillus flavus*, *Fusarium solani*, *Exophilia jeanselmei*, *Paecilomyces lilacinus*, *Acremonium curvulum*, *Alternaria infectoria*, *Scopulariopsis brevicaulis*, and *Cylindrocarpon tonkinese* [74, 85].

Medical management of post-traumatic endophthalmitis includes the use of prophylactic antibiotics following globe trauma as well as specific antimicrobial therapy of suspected and confirmed cases of endophthalmitis. Antibiotic prophylaxis includes antimicrobials administered prior to any clinical manifestations suggesting endophthalmitis [50]. Despite no widely accepted guidelines, prophylactic antibiotics are often recommended for patients with penetrating or perforating injuries to the globe [49, 56, 74, 116, 117]. Broad-spectrum, systemic, topical, and subconjunctival antibiotics are typically used in these cases. The contribution of systemic prophylactic measures to prevention of endophthalmitis is uncertain; however, systemic antibiotics are typically initiated if there is suspected or confirmed penetrating or perforating injury to the globe [49, 83, 118, 119]. Prophylactic intravenous systemic antibiotics are commonly used following combat injuries and civilian mass casualty events due to the presence of non-ocular injuries [120–124]. The choice of specific systemic antibiotics is based upon a number of factors, including the patient's overall condition, setting of the trauma, wound contamination, and presence of intraocular foreign bodies. Intravenous vancomycin is often used with ceftazidime to provide broad-spectrum antibiotic prophylaxis [49, 56, 74, 125]. Vancomycin is effective against most gram-positive organisms, including *Bacillus* species, while ceftazidime provides good gram-negative coverage. For patients with normal renal function, vancomycin 1 g every 12 hours and ceftazidime 1 g every 8 hours is a commonly used regimen [49, 126]. Clindamycin 300 mg every 8 hours may be used in patients allergic to vancomycin, while a fluoroquinolone may be substituted for ceftazidime in patients with a penicillin allergy. The optimal duration of intravenous antibiotic administration has not been established and varies among reports from 2 to 10 days [49, 56, 127].

Topical and subconjunctival antibiotics are begun after primary repair of open-globe injury. Despite no specific guidelines for the use of specific topical and/or subconjunctival antibiotics following surgical repair of globe trauma, most studies describe the use of at least topical antibiotics following primary repair of the wound. Various non-fortified topical antibiotics have been used following repair of globe trauma, including ciprofloxacin, moxifloxacin, levofloxacin, tobramycin, gentamicin, or chloramphenicol [73, 100, 118, 128, 129]. Prophylactic subconjunctival antibiotics include cefazolin, gentamicin, clindamycin, vancomycin, and ceftazidime [100, 116, 119, 130]. The use of prophylactic intravitreal antibiotics at the time of surgical repair is controversial, yet patients at high risk for traumatic endophthalmitis may benefit from prophylactic intravitreal antibiotics [49, 50, 83, 90, 106, 109, 117, 131, 132]. Criteria for these high-risk cases varies but probably should include patients with intraocular foreign bodies, lens disruption, injury in a rural setting, and wounds contaminated by vegetable or organic matter [56]. Intravitreal vancomycin 1 mg/0.1 ml and ceftazidime 2.25 mg/0.1 ml are most commonly used for prophylaxis in these situations [51, 56, 83, 125, 129, 131].

Management of cases of suspected traumatic endophthalmitis begins with obtaining specimens for culture and sensitivities as described previously. Broad-spectrum antibiotics are started immediately thereafter while awaiting the results of cultures. Empiric intravenous antibiotics should have good intravitreal penetration and sensitivity against the spectrum of bacteria commonly associated with ocular trauma. Sensitivity against *Bacillus* species should also be considered when choosing systemic antibiotics due to the high incidence of *Bacillus* species endophthalmitis following trauma [52, 56, 72, 74, 107, 116]. Intravenous vancomycin 1 g every 12 hours and ceftazidime 1 g every 8 hours provide good coverage against most organisms encountered following globe trauma including *Bacillus* spe-

cies [56, 74, 98, 126]. Intravenous clindamycin 300 mg every 8 hours may be used if vancomycin is contraindicated; especially if there is a clinical suspicion of *Bacillus* species infection [74, 105]. Several fluoroquinolones including ciprofloxacin, levofloxacin, and moxifloxacin achieve good aqueous and vitreous levels and may be useful alternative agents, but there are limited data regarding their use in traumatic endophthalmitis [49, 73, 101, 119, 133]. Intravenous voriconazole is recommended for all patients with post-traumatic fungal endophthalmitis based upon its coverage against *Candida* and *Aspergillus* species [74, 82, 134].

Intravitreal antibiotics are administered immediately after intraocular specimens are obtained. Initial antibiotic therapy should be broad spectrum to cover both gram-positive and gram-negative organisms. In most cases of trauma, infection with *Bacillus* species should be considered when selecting antibiotics [56, 74]. Intravitreal vancomycin 1 mg/0.1 ml and ceftazidime 2.25 mg/0.1 ml are typically used to provide broad-spectrum coverage following trauma [56, 126, 128]. In patients with suspected fungal endophthalmitis, intravitreal amphotericin B 5 μg/0.1 ml or voriconazole 100 μg/0.1 ml is typically used as initial therapy [56, 135]. Intravitreal corticosteroids may be considered in the management of post-traumatic bacterial endophthalmitis; however, their use remains somewhat controversial. On the contrary, intravitreal corticosteroids should not be used in cases of suspected or proven fungal endophthalmitis [56]. Corticosteroids modulate the immune response and limit tissue damage by inflammatory cells as well as bacterial toxins [136–139]. Nevertheless, it is important to recognize that intravitreal corticosteroids may exacerbate the infection if the infecting organism is not sensitive to the empiric intravitreal antibiotics [74]. Dexamethasone 0.4 mg/0.1 ml is the most commonly used intravitreal corticosteroid in cases of bacterial endophthalmitis [140, 141].

Topical broad-spectrum antibiotics are typically used in combination with intravitreal antibiotics while awaiting culture results. The most commonly used regimen in cases of suspected traumatic endophthalmitis is fortified vancomycin hydrochloride (50 mg/ml) with ceftazidime (100 mg/ml) [74]. Both of these drugs are administered every hour. An alternative combination consists of fortified gentamicin or tobramycin 14 mg/ml with fortified cefazolin 50 mg/ml. Topical fluoroquinolones including ciprofloxacin 0.3% or moxifloxacin 0.3% are additional options for initial therapy [74]. Topical antibiotics can be modified following results of microbiology cultures based upon sensitivities of the isolated organism. Topical corticosteroids as well as cycloplegics are also frequently administered in conjunction with antibiotic therapy [56].

Subconjunctival antibiotics are often used in the management of endophthalmitis. In patients with suspected traumatic endophthalmitis, subconjunctival vancomycin 25 mg and ceftazidime 100 mg are commonly used [56]. Other subconjunctival antibiotic options include tobramycin, gentamicin, or amikacin, although there are limited data regarding their use following globe trauma [116, 129, 142]. Subconjunctival injection of aminoglycosides should be done with extreme caution to avoid intraocular injection. Additionally, aminoglycosides should not be injected into the subconjunctival space if there is any evidence or suspicion of an open wound [126, 143].

If cultures identify a specific organism or multiple organisms, the sensitivities should be reviewed to determine the most appropriate antimicrobial therapy. In some cases, this may require switching to a different antimicrobial agent and/or discontinuation of broad-spectrum drugs. Repeat intravitreal injections may be necessary in some patients based upon culture results or those with no improvement or worsening inflammation. Repeat intravitreal antibiotics or antifungals might also be considered in eyes with highly virulent or resistant organisms such as *Bacillus* species [56, 144].

Vitrectomy may be utilized in selected cases of traumatic endophthalmitis. Pars plana vitrectomy is often necessary for management of retinal detachment, intraocular foreign bodies, and posterior dislocated lens material. Recommendations regarding timing of vitrectomy in patients with

intraocular foreign bodies differ but some surgeons advocate early removal of foreign bodies to avoid complications such as endophthalmitis and proliferative vitreoretinopathy [53, 79]. On the other hand, recent studies suggest that delayed removal may not increase the risk of endophthalmitis, especially if patients receive prophylactic antibiotics prior to surgery [53, 73, 78–80, 145]. Pars plana vitrectomy may also be useful in patients with severe inflammation and no response to intravitreal antibiotics within 48 hours or marked worsening of inflammation after 24 hours following initial therapy [50, 72, 74]. Possible benefits of vitrectomy in these cases are obtaining additional vitreous for culture, reduction in the pathogen load, and partial clearing of vitreous opacities [75, 97, 146]. However, there are no widely accepted recommendations regarding timing, surgical techniques, or use of antibiotics in the irrigating solutions during vitrectomy in patients with traumatic endophthalmitis. Bhagat and coworkers suggest a limited vitrectomy in these situations based upon the suboptimal view of the posterior segment and high risk for retinal tears and detachment associated with the severe inflammation [74].

References

1. Duke-Elder S, MacFaul PA. Concussions and contusions. In: Duke-Elder S, editor. System of ophthalmology, Vol. XIV, Injuries, Part 1: Mechanical injuries. St. Louis: Mosby; 1972. p. 63–149.
2. Duma SM, Ng TP, Kennedy EA, Stitzel JD, Herring IP, Kuhn F. Determination of significant parameters for eye injury risk from projectiles. J Trauma. 2005;59(4):960–4.
3. Ramstead C, Ng M, Rudnisky CJ. Ocular injuries associated with Airsoft guns: a case series. Can J Ophthalmol. 2008;43(5):584–7.
4. Canavan YM, Archer DB. Anterior segment consequences of blunt ocular injury. Br J Ophthalmol. 1982;66(9):549–55.
5. Schein OD, Hibberd PL, Shingleton BJ, Kunzweiler T, Frambach DA, Seddon JM, et al. The spectrum and burden of ocular injury. Ophthalmology. 1988;95(3):300–5.
6. Macewen CJ. Eye injuries: a prospective survey of 5671 cases. Br J Ophthalmol. 1989;73(11):888–94.
7. Capao Filipe JA. Modern sports eye injuries. Br J Ophthalmol. 2003;87(11):1336–9.
8. Morley MG, Nguyen JK, Heier JS, Shingleton BJ, Pasternak JF, Bower KS. Blast eye injuries: a review for first responders. Disaster Med Public Health Prep. 2010;4(2):154–60.
9. Mines M, Thach A, Mallonee S, Hildebrand L, Shariat S. Ocular injuries sustained by survivors of the Oklahoma City bombing. Ophthalmology. 2000;107(5):837–43.
10. Quere MA, Bouchat J, Cornand G. Ocular blast injuries. Am J Ophthalmol. 1969;67(1):64–9.
11. Beiran I, Miller B. Pure ocular blast injury. Am J Ophthalmol. 1992;114(4):504–5.
12. Wong TY, Seet MB, Ang CL. Eye injuries in twentieth century warfare: a historical perspective. Surv Ophthalmol. 1997;41(6):433–59.
13. Abbotts R, Harrison SE, Cooper GL. Primary blast injuries to the eye: a review of the evidence. J R Army Med Corps. 2007;153(2):119–23.
14. Blanch RJ, Bindra MS, Jacks AS, Scott RA. Ophthalmic injuries in British armed forces in Iraq and Afghanistan. Eye (Lond). 2011;25(2):218–23.
15. Scott R. The injured eye. Philos Trans R Soc Lond Ser B Biol Sci. 2011;366(1562):251–60.
16. Banitt MR, Malta JB, Mian SI, Soong HK. Rupture of anterior lens capsule from blunt ocular injury. J Cataract Refract Surg. 2009;35(5):943–5.
17. Maloney WF, Colvard M, Bourne WM, Gardon R. Specular microscopy of traumatic posterior annular keratopathy. Arch Ophthalmol. 1979;97(9):1647–50.
18. Cibis GW, Weingeist TA, Krachmer JH. Traumatic corneal endothelial rings. Arch Ophthalmol. 1978;96(3):485–8.
19. De Leon-Ortega JE, Girkin CA. Ocular trauma-related glaucoma. Ophthalmol Clin N Am. 2002;15(2):215–23.
20. Milder E, Davis K. Ocular trauma and glaucoma. Int Ophthalmol Clin. 2008;48(4):47–64.
21. Deramo VA, Shah GK, Baumal CR, Fineman MS, Corrĕa ZM, Benson WE, et al. The role of ultrasound biomicroscopy in ocular trauma. Trans Am Ophthalmol Soc. 1998;96:355–65; discussion 365–7.
22. Deramo VA, Shah GK, Baumal CR, Fineman MS, Corrêa ZM, Benson WE, et al. Ultrasound biomicroscopy as a tool for detecting and localizing occult foreign bodies after ocular trauma. Ophthalmology. 1999;106(2):301–5.
23. Patel SN, Langer PD, Zarbin MA, Bhagat N. Diagnostic value of clinical examination and radiographic imaging in identification of intraocular foreign bodies in open globe injury. Eur J Ophthalmol. 2012;22(2):259–68.
24. Moisseiev E, Last D, Goez D, Barak A, Mardor Y. Magnetic resonance imaging and computed tomography for the detection and characterization of nonmetallic intraocular foreign bodies. Retina. 2015;35(1):82–94.
25. Marak GE. Phacoanaphylactic endophthalmitis. Surv Ophthalmol. 1992;36(5):325–39.
26. Gery I, Nussenblatt R, BenEzra D. Dissociation between humoral and cellular immune responses

to lens antigens. Invest Ophthalmol Vis Sci. 1981;20(1):32–9.
27. Goldschmidt L, Goldbaum M, Walker SM, Weigle WO. The immune response to homologous lens crystallin. I. Antibody production after lens injury. J Immunol. 1982;129(4):1652–7.
28. Lai JC, Lobanoff MC, Fukushima A, Wawrousek EF, Chan CC, Whitcup SM, Gery I. Uveitis induced by lymphocytes sensitized against a transgenically expressed lens protein. Invest Ophthalmol Vis Sci. 1999;40(11):2735–9.
29. Perlman EM, Albert DM. Clinically unsuspected phacoanaphylaxis after ocular trauma. Arch Ophthalmol. 1977;95(2):244–6.
30. Thach AB, Marak GE, McLean IW, Green WR. Phacoanaphylactic endophthalmitis: a clinicopathologic review. Int Ophthalmol. 1991;15(4):271–9.
31. Zabriskie NA, Hwang IP, Ramsey JF, Crandall AS. Anterior lens capsule rupture caused by air bag trauma. Am J Ophthalmol. 1997;123(6):832–3.
32. Dezhagah H. Circular anterior lens capsule rupture caused by blunt ocular trauma. Middle East Afr J Ophthalmol. 2010;17(1):103–5.
33. Dansey-Browning GC. The value of ophthalmic treatment in the field. Br J Ophthalmol. 1944;28(2):87–97.
34. Chandler PA. Problems in the diagnosis and treatment of lens-induced uveitis and glaucoma. AMA Arch Ophthalmol. 1958;60(5):828–41.
35. Muga R, Maul E. The management of lens damage in perforating corneal lacerations. Br J Ophthalmol. 1978;62(11):784–7.
36. Lamkin JC, Azar DT, Mead MD, Volpe NJ. Simultaneous corneal laceration repair, cataract removal, and posterior chamber intraocular lens implantation. Am J Ophthalmol. 1992;113(6):626–31.
37. Moisseiev J, Segev F, Harizman N, Arazi T, Rotenstreich Y, Assia EI. Primary cataract extraction and intraocular lens implantation in penetrating ocular trauma. Ophthalmology. 2001;108(6):1099–103.
38. Tanito M, Kaidzu S, Katsube T, Nonoyama S, Takai Y, Ohira A. Diagnostic Western blot for lens-specific proteins in aqueous fluid after traumatic lens-induced uveitis. Jpn J Ophthalmol. 2009;53(4):436–9.
39. Inazumi K, Gentile RC, Lee KY, Ishikawa H, McCormick SA, Liebmann JM, Ritch R. Ultrasound biomicroscopic diagnosis of cyclitic membranes. Am J Ophthalmol. 2001;131(4):446–50.
40. Chan CC, Fujikawa LS, Rodrigues MM, Stevens G, Nussenblatt RB. Immunohistochemistry and electron microscopy of cyclitic membrane. Report of a case. Arch Ophthalmol. 1986;104(7):1040–5.
41. Petersen K, Riddle MS, Danko JR, Blazes DL, Hayden R, Tasker SA, Dunne JR. Trauma-related infections in battlefield casualties from Iraq. Ann Surg. 2007;245(5):803–11.
42. Murray CK. Infectious disease complications of combat-related injuries. Crit Care Med. 2008;36(7 Suppl):S358–64.
43. Murray CK. Epidemiology of infections associated with combat-related injuries in Iraq and Afghanistan. J Trauma. 2008;64(3 Suppl):S232–8.
44. Murray CK, Hinkle MK, Yun HC. History of infections associated with combat-related injuries. J Trauma. 2008;64(3 Suppl):S221–31.
45. Kluger Y. Bomb explosions in acts of terrorism--detonation, wound ballistics, triage and medical concerns. Isr Med Assoc J. 2003;5(4):235–40.
46. Kluger Y, Peleg K, Daniel-Aharonson L, Mayo A, Israeli Trauma Group. The special injury pattern in terrorist bombings. J Am Coll Surg. 2004;199(6):875–9.
47. Bartels SA, VanRooyen MJ. Medical complications associated with earthquakes. Lancet. 2012;379(9817):748–57.
48. Keven K, Ates K, Sever MS, Yenicesu M, Canbakan B, Arinsoy T, et al. Infectious complications after mass disasters: the Marmara earthquake experience. Scand J Infect Dis. 2003;35(2):110–3.
49. Andreoli CM, Andreoli MT, Kloek CE, Ahuero AE, Vavvas D, Durand ML. Low rate of endophthalmitis in a large series of open globe injuries. Am J Ophthalmol. 2009;147(4):601–608.e2.
50. Essex RW, Yi Q, Charles PG, Allen PJ. Post-traumatic endophthalmitis. Ophthalmology. 2004;111(11):2015–22.
51. Boldt HC, Pulido JS, Blodi CF, Folk JC, Weingeist TA. Rural endophthalmitis. Ophthalmology. 1989;96(12):1722–6.
52. Thompson JT, Parver LM, Enger CL, Mieler WF, Liggett PE. Infectious endophthalmitis after penetrating injuries with retained intraocular foreign bodies. National eye trauma system. Ophthalmology. 1993;100(10):1468–74.
53. Jonas JB, Knorr HL, Budde WM. Prognostic factors in ocular injuries caused by intraocular or retrobulbar foreign bodies. Ophthalmology. 2000;107(5):823–8.
54. Duch-Samper AM, Menezo JL, Hurtado-Sarrió M. Endophthalmitis following penetrating eye injuries. Acta Ophthalmol Scand. 1997;75(1):104–6.
55. Verbraeken H, Rysselaere M. Post-traumatic endophthalmitis. Eur J Ophthalmol. 1994;4(1):1–5.
56. Ahmed Y, Schimel AM, Pathengay A, Colyer MH, Flynn HW. Endophthalmitis following open-globe injuries. Eye (Lond). 2012;26(2):212–7.
57. Ahmed SA, Zaki RG. Forensic analysis of ocular injuries during the 2011 revolution in Egypt. Forensic Sci Int. 2013;233(1–3):348–54.
58. Muzaffar W, Khan MD, Akbar MK, Malik AM, Durrani OM. Mine blast injuries: ocular and social aspects. Br J Ophthalmol. 2000;84(6):626–30.
59. Parke DW, Pathengay A, Flynn HW, Albini T, Schwartz SG. Risk factors for endophthalmitis and retinal detachment with retained intraocular foreign bodies. J Ophthalmol. 2012;2012:758526.
60. Treister G. Ocular casualties in the six-day war. Am J Ophthalmol. 1969;68(4):669–75.
61. Anderson WD. Prophylactic antibiotics and endophthalmitis in Vietnam. Am J Ophthalmol. 1973;75(3):481–5.

62. Lashkari K, Lashkari MH, Kim AJ, Crane WG, Jalkh AE. Combat-related eye trauma: a review of 5,320 cases. Int Ophthalmol Clin. 1995;35(1):193–203.
63. Thach AB, Ward TP, Dick JS, Bauman WC, Madigan WP, Goff MJ, Thordsen JE. Intraocular foreign body injuries during Operation Iraqi Freedom. Ophthalmology. 2005;112(10):1829–33.
64. Weichel ED, Colyer MH, Ludlow SE, Bower KS, Eiseman AS. Combat ocular trauma visual outcomes during operations Iraqi and Enduring Freedom. Ophthalmology. 2008;115(12):2235–45.
65. Yang CS, Lu CK, Lee FL, Hsu WM, Lee YF, Lee SM. Treatment and outcome of traumatic endophthalmitis in open globe injury with retained intraocular foreign body. Ophthalmologica. 2010;224(2):79–85.
66. Klein JS, Weigelt JA. Disaster management. Lessons learned. Surg Clin North Am. 1991;71(2):257–66.
67. Singer AJ, Singer AH, Halperin P, Kaspi G, Assaf J. Medical lessons from terror attacks in Israel. J Emerg Med. 2007;32(1):87–92.
68. McAlister CN, Murray TJ, Lakosha H, Maxner CE. The Halifax disaster (1917): eye injuries and their care. Br J Ophthalmol. 2007;91(6):832–5.
69. Thach AB, Johnson AJ, Carroll RB, Huchun A, Ainbinder DJ, Stutzman RD, et al. Severe eye injuries in the war in Iraq, 2003-2005. Ophthalmology. 2008;115(2):377–82.
70. Butler FK, Blackbourne LH. Battlefield trauma care then and now: a decade of tactical combat casualty care. J Trauma Acute Care Surg. 2012;73(6 Suppl 5):S395–402.
71. Sobaci G, Mutlu FM, Bayer A, Karagül S, Yildirim E. Deadly weapon-related open-globe injuries: outcome assessment by the ocular trauma classification system. Am J Ophthalmol. 2000;129(1):47–53.
72. Brinton GS, Topping TM, Hyndiuk RA, Aaberg TM, Reeser FH, Abrams GW. Posttraumatic endophthalmitis. Arch Ophthalmol. 1984;102(4):547–50.
73. Colyer MH, Weber ED, Weichel ED, Dick JS, Bower KS, Ward TP, Haller JA. Delayed intraocular foreign body removal without endophthalmitis during Operations Iraqi Freedom and Enduring Freedom. Ophthalmology. 2007;114(8):1439–47.
74. Bhagat N, Nagori S, Zarbin M. Post-traumatic infectious endophthalmitis. Surv Ophthalmol. 2011;56(3):214–51.
75. Cornut PL, Youssef el B, Bron A, Thuret G, Gain P, Burillon C, et al. A multicentre prospective study of post-traumatic endophthalmitis. Acta Ophthalmol. 2013;91(5):475–82.
76. Das T, Kunimoto DY, Sharma S, Jalali S, Majji AB, Nagaraja Rao T, et al. Relationship between clinical presentation and visual outcome in postoperative and posttraumatic endophthalmitis in south central India. Indian J Ophthalmol. 2005;53(1):5–16.
77. Nicoară SD, Irimescu I, Călinici T, Cristian C. Outcome and prognostic factors for traumatic endophthalmitis over a 5-year period. J Ophthalmol. 2014;2014:747015.
78. Wani VB, Al-Ajmi M, Thalib L, Azad RV, Abul M, Al-Ghanim M, Sabti K. Vitrectomy for posterior segment intraocular foreign bodies: visual results and prognostic factors. Retina. 2003;23(5):654–60.
79. Erakgun T, Egrilmez S. Prognostic factors in vitrectomy for posterior segment intraocular foreign bodies. J Trauma. 2008;64(4):1034–7.
80. Choovuthayakorn J, Hansapinyo L, Ittipunkul N, Patikulsila D, Kunavisarut P. Predictive factors and outcomes of posterior segment intraocular foreign bodies. Eye (Lond). 2011;25(12):1622–6.
81. Wykoff CC, Flynn HW, Miller D, Scott IU, Alfonso EC. Exogenous fungal endophthalmitis: microbiology and clinical outcomes. Ophthalmology. 2008;115(9):1501–7, 1507.e1–2
82. Silva RA, Sridhar J, Miller D, Wykoff CC, Flynn HW. Exogenous fungal endophthalmitis: an analysis of isolates and susceptibilities to antifungal agents over a 20-year period (1990–2010). Am J Ophthalmol. 2015;159(2):257–264.e1.
83. Reynolds DS, Flynn HW. Endophthalmitis after penetrating ocular trauma. Curr Opin Ophthalmol. 1997;8(3):32–8.
84. Thompson WS, Rubsamen PE, Flynn HW, Schiffman J, Cousins SW. Endophthalmitis after penetrating trauma. Risk factors and visual acuity outcomes. Ophthalmology. 1995;102(11):1696–701.
85. Gupta A, Srinivasan R, Kaliaperumal S, Saha I. Post-traumatic fungal endophthalmitis--a prospective study. Eye (Lond). 2008;22(1):13–7.
86. Abu el-Asrar AM, al-Amro SA, al-Mosallam AA, al-Obeidan S. Post-traumatic endophthalmitis: causative organisms and visual outcome. Eur J Ophthalmol. 1999;9(1):21–31.
87. Han DP, Wisniewski SR, Kelsey SF, Doft BH, Barza M, Pavan PR. Microbiologic yields and complication rates of vitreous needle aspiration versus mechanized vitreous biopsy in the endophthalmitis vitrectomy study. Retina. 1999;19(2):98–102.
88. Allansmith MR, Skaggs C, Kimura SJ. Anterior chamber paracentesis. Diagnostic value in postoperative endophthalmitis. Arch Ophthalmol. 1970;84(6):745–8.
89. Forster RK. Endophthalmitis. Diagnostic cultures and visual results. Arch Ophthalmol. 1974;92(5):387–92.
90. Peyman GA, Carroll CP, Raichand M. Prevention and management of traumatic endophthalmitis. Ophthalmology. 1980;87(4):320–4.
91. Rowsey JJ, Newsom DL, Sexton DJ, Harms WK. Endophthalmitis: current approaches. Ophthalmology. 1982;89(9):1055–66.
92. Affeldt JC, Flynn HW, Forster RK, Mandelbaum S, Clarkson JG, Jarus GD. Microbial endophthalmitis resulting from ocular trauma. Ophthalmology. 1987;94(4):407–13.
93. Baron EJ, Miller JM, Weinstein MP, Richter SS, Gilligan PH, Thomson RB, et al. A guide to utilization of the microbiology laboratory for diagnosis of infectious diseases: 2013 recommendations by the Infectious Diseases Society of America (IDSA) and

the American Society for Microbiology (ASM)(a). Clin Infect Dis. 2013;57(4):e22–e121.
94. Thomas PA. Current perspectives on ophthalmic mycoses. Clin Microbiol Rev. 2003;16(4):730–97.
95. Nobe JR, Gomez DS, Liggett P, Smith RE, Robin JB. Post-traumatic and postoperative endophthalmitis: a comparison of visual outcomes. Br J Ophthalmol. 1987;71(8):614–7.
96. Jindal A, Moreker MR, Pathengay A, Khera M, Jalali S, Majji A, et al. Polymicrobial endophthalmitis: prevalence, causative organisms, and visual outcomes. J Ophthalmic Inflamm Infect. 2013;3(1):6.
97. Alfaro DV, Roth D, Liggett PE. Posttraumatic endophthalmitis. Causative organisms, treatment, and prevention. Retina. 1994;14(3):206–11.
98. Chhabra S, Kunimoto DY, Kazi L, Regillo CD, Ho AC, Belmont J, et al. Endophthalmitis after open globe injury: microbiologic spectrum and susceptibilities of isolates. Am J Ophthalmol. 2006;142(5):852–4.
99. Rubsamen PE, Cousins SW, Martinez JA. Impact of cultures on management decisions following surgical repair of penetrating ocular trauma. Ophthalmic Surg Lasers. 1997;28(1):43–9.
100. Sabaci G, Bayer A, Mutlu FM, Karagül S, Yildirim E. Endophthalmitis after deadly-weapon-related open-globe injuries: risk factors, value of prophylactic antibiotics, and visual outcomes. Am J Ophthalmol. 2002;133(1):62–9.
101. Kunimoto DY, Das T, Sharma S, Jalali S, Majji AB, Gopinathan U, et al. Microbiologic spectrum and susceptibility of isolates: part II. Posttraumatic endophthalmitis. Endophthalmitis research group. Am J Ophthalmol. 1999;128(2):242–4.
102. David DB, Kirkby GR, Noble BA. Bacillus cereus endophthalmitis. Br J Ophthalmol. 1994;78(7):577–80.
103. Long C, Liu B, Xu C, Jing Y, Yuan Z, Lin X. Causative organisms of post-traumatic endophthalmitis: a 20-year retrospective study. BMC Ophthalmol. 2014;14:34.
104. Miller JJ, Scott IU, Flynn HW, Smiddy WE, Murray TG, Berrocal A, Miller D. Endophthalmitis caused by bacillus species. Am J Ophthalmol. 2008;145(5):883–8.
105. Schemmer GB, Driebe WT. Posttraumatic bacillus cereus endophthalmitis. Arch Ophthalmol. 1987;105(3):342–4.
106. Zhang Y, Zhang MN, Jiang CH, Yao Y, Zhang K. Endophthalmitis following open globe injury. Br J Ophthalmol. 2010;94(1):111–4.
107. O'Day DM, Smith RS, Gregg CR, Turnbull PC, Head WS, Ives JA, Ho PC. The problem of bacillus species infection with special emphasis on the virulence of bacillus cereus. Ophthalmology. 1981;88(8):833–8.
108. Davey RT, Tauber WB. Posttraumatic endophthalmitis: the emerging role of bacillus cereus infection. Rev Infect Dis. 1987;9(1):110–23.
109. Lieb DF, Scott IU, Flynn HW, Miller D, Feuer WJ. Open globe injuries with positive intraocular cultures: factors influencing final visual acuity outcomes. Ophthalmology. 2003;110(8):1560–6.
110. Wiskur BJ, Robinson ML, Farrand AJ, Novosad BD, Callegan MC. Toward improving therapeutic regimens for bacillus endophthalmitis. Invest Ophthalmol Vis Sci. 2008;49(4):1480–7.
111. Bottone EJ. Bacillus cereus, a volatile human pathogen. Clin Microbiol Rev. 2010;23(2):382–98.
112. Berrocal AM, Scott IU, Miller D, Flynn HW. Endophthalmitis caused by moraxella species. Am J Ophthalmol. 2001;132(5):788–90.
113. Lai TY, Kwok AK, Fung KS, Chan W-M, Fan DS, Lam DS. Stenotrophomonas maltophilia endophthalmitis after penetrating injury by a wooden splinter. Eye. 2001;15(3):353–4.
114. Patton N. Post-traumatic endophthalmitis caused by xanthomonas maltophilia. Eye (Lond). 2001;15(Pt 6):801–2.
115. Pflugfelder SC, Flynn HW, Zwickey TA, Forster RK, Tsiligianni A, Culbertson WW, Mandelbaum S. Exogenous fungal endophthalmitis. Ophthalmology. 1988;95(1):19–30.
116. Parrish CM, O'Day DM. Traumatic endophthalmitis. Int Ophthalmol Clin. 1987;27(2):112–9.
117. Mittra RA, Mieler WF. Controversies in the management of open-globe injuries involving the posterior segment. Surv Ophthalmol. 1999;44(3):215–25.
118. Ariyasu RG, Kumar S, LaBree LD, Wagner DG, Smith RE. Microorganisms cultured from the anterior chamber of ruptured globes at the time of repair. Am J Ophthalmol. 1995;119(2):181–8.
119. Colyer MH, Chun DW, Bower KS, Dick JS, Weichel ED. Perforating globe injuries during operation Iraqi Freedom. Ophthalmology. 2008;115(11):2087–93.
120. Hospenthal DR, Murray CK, Andersen RC, Blice JP, Calhoun JH, Cancio LC, et al. Guidelines for the prevention of infection after combat-related injuries. J Trauma. 2008;64(3 Suppl):S211–20.
121. Turégano-Fuentes F, Caba-Doussoux P, Jover-Navalón JM, Martín-Pérez E, Fernández-Luengas D, Díez-Valladares L, et al. Injury patterns from major urban terrorist bombings in trains: the Madrid experience. World J Surg. 2008;32(6):1168–75.
122. Aschkenasy-Steuer G, Shamir M, Rivkind A, Mosheiff R, Shushan Y, Rosenthal G, et al. Clinical review: the Israeli experience: conventional terrorism and critical care. Crit Care. 2005;9(5):490–9.
123. Yang C, Wang HY, Zhong HJ, Zhou L, Jiang DM, Du DY, et al. The epidemiological analyses of trauma patients in Chongqing teaching hospitals following the Wenchuan earthquake. Injury. 2009;40(5):488–92.
124. Miskin IN, Nir-Paz R, Block C, Merin O, Burshtein S, Pirogovsky S, et al. Antimicrobial therapy for wound infections after catastrophic earthquakes. N Engl J Med. 2010;363(26):2571–3.

125. Lorch A, Sobrin L. Prophylactic antibiotics in posttraumatic infectious endophthalmitis. Int Ophthalmol Clin. 2013;53(4):167–76.
126. Kresloff MS, Castellarin AA, Zarbin MA. Endophthalmitis. Surv Ophthalmol. 1998;43(3): 193–224.
127. Faghihi H, Hajizadeh F, Esfahani MR, Rasoulinejad SA, Lashay A, Mirshahi A, et al. Posttraumatic endophthalmitis: report no. 2. Retina. 2012;32(1):146–51.
128. Narang S, Gupta V, Gupta A, Dogra MR, Pandav SS, Das S. Role of prophylactic intravitreal antibiotics in open globe injuries. Indian J Ophthalmol. 2003;51(1):39–44.
129. Soheilian M, Rafati N, Mohebbi MR, Yazdani S, Habibabadi HF, Feghhi M, et al. Prophylaxis of acute posttraumatic bacterial endophthalmitis: a multicenter, randomized clinical trial of intraocular antibiotic injection, report 2. Arch Ophthalmol. 2007;125(4):460–5.
130. Mieler WF, Ellis MK, Williams DF, Han DP. Retained intraocular foreign bodies and endophthalmitis. Ophthalmology. 1990;97(11):1532–8.
131. Seal DV, Kirkness CM. Criteria for intravitreal antibiotics during surgical removal of intraocular foreign bodies. Eye (Lond). 1992;6(Pt 5):465–8.
132. Meredith TA. Posttraumatic endophthalmitis. Arch Ophthalmol. 1999;117(4):520–1.
133. Hariprasad SM, Shah GK, Mieler WF, Feiner L, Blinder KJ, Holekamp NM, et al. Vitreous and aqueous penetration of orally administered moxifloxacin in humans. Arch Ophthalmol. 2006;124(2):178–82.
134. Hariprasad SM, Mieler WF, Holz ER, Gao H, Kim JE, Chi J, Prince RA. Determination of vitreous, aqueous, and plasma concentration of orally administered voriconazole in humans. Arch Ophthalmol. 2004;122(1):42–7.
135. Kernt M, Neubauer AS, De Kaspar HM, Kampik A. Intravitreal voriconazole: in vitro safety-profile for fungal endophthalmitis. Retina. 2009;29(3):362–70.
136. Meredith TA, Aguilar HE, Miller MJ, Gardner SK, Trabelsi A, Wilson LA. Comparative treatment of experimental staphylococcus epidermidis endophthalmitis. Arch Ophthalmol. 1990;108(6):857–60.
137. Schulman JA, Peyman GA. Intravitreal corticosteroids as an adjunct in the treatment of bacterial and fungal endophthalmitis. A review. Retina. 1992;12(4):336–40.
138. Park SS, Samiy N, Ruoff K, D'Amico DJ, Baker AS. Effect of intravitreal dexamethasone in treatment of pneumococcal endophthalmitis in rabbits. Arch Ophthalmol. 1995;113(10):1324–9.
139. Kernt M, Kampik A. Endophthalmitis: pathogenesis, clinical presentation, management, and perspectives. Clin Ophthalmol. 2010;4:121–35.
140. Das T, Jalali S, Gothwal VK, Sharma S, Naduvilath TJ. Intravitreal dexamethasone in exogenous bacterial endophthalmitis: results of a prospective randomised study. Br J Ophthalmol. 1999;83(9): 1050–5.
141. Albrecht E, Richards JC, Pollock T, Cook C, Myers L. Adjunctive use of intravitreal dexamethasone in presumed bacterial endophthalmitis: a randomised trial. Br J Ophthalmol. 2011;95(10):1385–8.
142. Smiddy WE, Smiddy RJ, Ba'Arath B, Flynn HW, Murray TG, Feuer WJ, Miller D. Subconjunctival antibiotics in the treatment of endophthalmitis managed without vitrectomy. Retina. 2005;25(6): 751–8.
143. Judson PH. Aminoglycoside macular toxicity after subconjunctival injection. Case report. Arch Ophthalmol. 1989;107(9):1282–3.
144. Shaarawy A, Grand MG, Meredith TA, Ibanez HE. Persistent endophthalmitis after intravitreal antimicrobial therapy. Ophthalmology. 1995;102(3):382–7.
145. Hutton WL, Fuller DG. Factors influencing final visual results in severely injured eyes. Am J Ophthalmol. 1984;97(6):715–22.
146. Duch-Samper AM, Chaqués-Alepuz V, Menezo JL, Hurtado-Sarrió M. Endophthalmitis following open-globe injuries. Curr Opin Ophthalmol. 1998;9(3):59–65.

Part II

Special Considerations

13 Prehospital Care of Combat Eye Injuries

Frank K. Butler and Robert A. Mazzoli

Background

Despite comprising only 0.1% of the total body surface area, the incidence of casualties with ocular and ocular adnexal injuries as a percentage of the total number of combat casualties has historically ranged from 0.65% in the Crimean War to 13% in Desert Storm [1], a much higher incidence than would be expected given the small size of the organ. Similarly, eye and vision injuries exert an inordinate effect on combat readiness; it is a given that "If you can't see, you can't fight." Although recent advances in both ballistic eyewear and ophthalmic surgical care have dramatically reduced the incidence of severe vision loss or loss of the eye associated with these injuries, [2–5] the eye remains highly sensitive to injury. Eye injuries may be just one component of the complex polytrauma wounding pattern typical of modern combat, especially from high-explosive blast injuries [6–9]. Indeed, ocular injuries may occur in over 30% of combat cranial traumas; likewise, cranial trauma accompanies over 20% of modern combat eye injuries [10]. Over 40% of eye injuries may have concomitant extremity injuries [11]. Similarly, most combat ocular trauma represents ocular polytrauma, with multiple ocular structures involved, such as the lids, cornea, sclera, retina, optic nerve, ocular pressure regulating system, and orbit [11].

Because the eye is notoriously intolerant of injury—and of error—ocular injuries are best treated by ophthalmic surgeons and subspecialists who are expert in repair techniques. In reality, there is little definitive care that can be provided the ocular casualty in the preophthalmic zone of care by nonophthalmologists, and this should temper any desire to do so. But this does not mean ignore the eye. On the contrary, the first response and treatment afforded at the point of injury (POI) and at subsequent echelons set the foundation for any subsequent repair attempt and are vitally important. These first actions, largely aimed at minimizing subsequent damage and preventing early infection, are critical to the final visual outcome and can make the difference between an eye that is salvaged and an eye that is lost. In short, early actions can determine if the eye is "KIA" or if it "arrives alive" at the ophthalmic facility.

Understanding that ophthalmologists will probably not be deployed to every theater Combat

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Support Hospital/Role 3 but will more likely be a scarce theater-level asset, we will use the terms "preophthalmic" and "prehospital" rather interchangeably, denoting care given at any forward location where ophthalmologists are not assigned. More specifically, "prehospital" should be interpreted to mean first responder care provided in field conditions, whereas "preophthalmic" would mean care provided anywhere there is no ophthalmic support, including more established higher echelon facilities. This section will focus on the prehospital/preophthalmic care that should be undertaken for most significant eye injury types.

Shield and Ship

The modern battlefield contains an array of hazards that may result in a variety of combat eye injuries from both penetrating and nonpenetrating mechanisms, including all of the injury types in Table 13.1 [12–15]. Interestingly, but not surprisingly, many of these same injuries were identified in the high-explosive environments of the First and Second World Wars and other incidents in the past [16–20]. Increasingly, combat-style ocular injuries are being seen in domestic incidents, both intentional and accidental [6].

Table 13.1 Types of combat eye injuries

Penetrating globe injury and globe rupture
Blunt force/nonpenetrating trauma to the globe
Eyelid lacerations
Tissue avulsion
Blast injury
Corneal abrasions
Conjunctival and corneal foreign bodies
Internal ocular injury
Retinal detachment
Intraocular hemorrhage
Intraocular foreign bodies
Orbital fractures
Orbital hemorrhage and compartment syndrome
Optic nerve injury, avulsion
Chemical injury
Traumatic optic neuropathy and optic nerve avulsion
Burn
Crush injury
Laser eye injury

Fundamentally, unlike other wounds, where direct pressure to control bleeding is usually indicated, ocular injuries can be seriously aggravated by direct or indirect pressure and inadvertent additional trauma, leading to loss of vision or the eye. Consequently, the overriding and most important principle in the preophthalmic care of eye injuries is that casualties with known or suspected penetrating eye trauma should immediately have a rigid protective shield placed over the injured eye(s) and be evaluated and treated by an ophthalmologist as soon as possible ("*shield and ship*") (Fig. 13.1). Anything that rigidly vaults over the eye and orbit can provide effective protection. Commercially manufactured shields should be widely available, but field expedients such as simply replacing and securing the casualty's ballistic eye armor will also work. *Do not patch* the eye or place gauze bandages on the eye or under the shield (Fig. 13.2). Unless there is a need for immediate lifesaving or resuscitative surgery at a Role 2 medical treatment facility, casualties with eye injuries should be immediately evacuated to a Role 3 hospital where ophthalmic specialty care is available (Urgent, or equivalent, for arrival not later than 12 hours for open-globe injuries). However, providers at every stage must ensure a rigid eyeshield is in place prior to transfer.

Evaluation of Casualties with Eye Injuries

The first clues to the presence of a penetrating eye injury are provided by the mechanism of injury and a quick look at the face. In a casualty with "peppering" of the face from blast fragments who was not wearing ballistic eye protection at the time of injury, penetrating eye trauma should be strongly suspected. Even if the casualty was wearing eye protection, the possibility of serious or penetrating injury should be considered if the periocular wounding pattern is suggestive or if there is eye pain and/or vision loss, since the blast force may dislodge the protective eyewear and allow fragments to injure the eye. Any injury to the ocular area that would other-

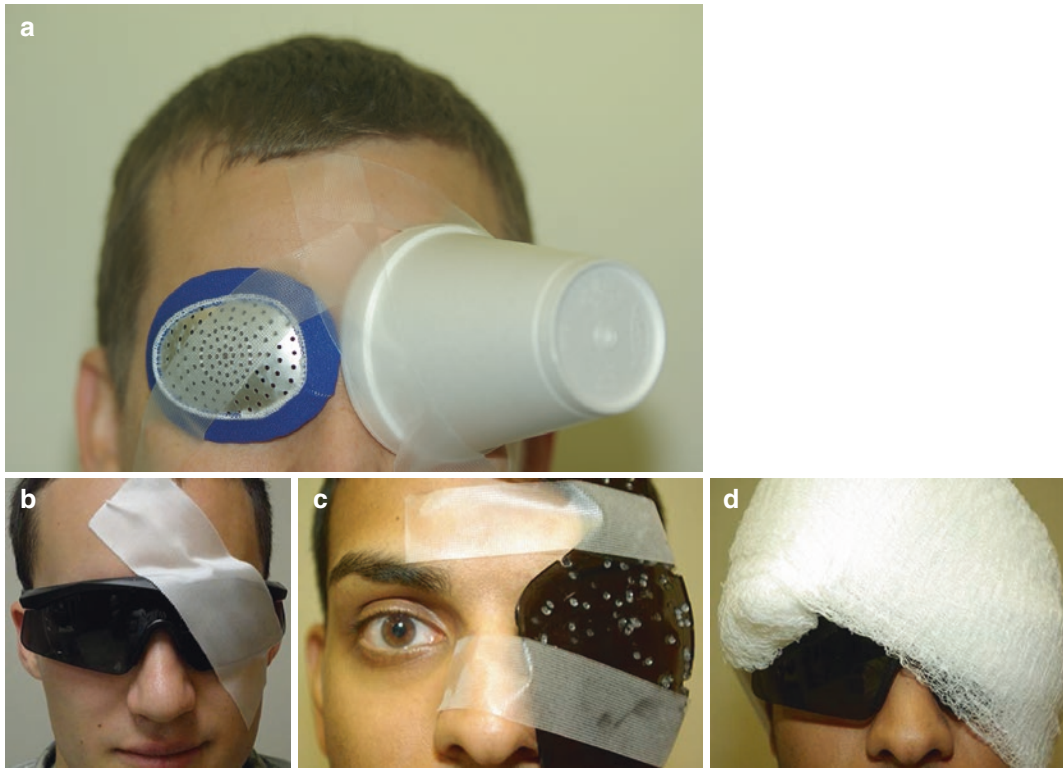

Fig. 13.1 Options for rigid eye shields. Protect all known or suspected penetrating eye injuries and significant non-penetrating injuries with a rigid eye shield that vaults over the eye without touching it. Do not patch the eye or place gauze or any other dressing over the eye or under the shield. Metal Fox shields (1a, right eye) are increasingly available in front-line aid kits such as the Individual First Aid Kit-II and the Joint First Aid Kit (IFAK-II/JFAK), Army 68W aid bags, Combat Lifesaver bags, and vehicular Warrior Aid and Litter Kits (WALK) kits. Metal shields can be molded, shaped, and contoured to accommodate swelling without touching the lids, just as other moldable splints are. If a commercial shield is unavailable, field expedients include replacing ballistic eye armor, even if moderately damaged (**b**, **c**); a cup (**a**, left eye); or anything that can vault cleanly over the eye (from the brow to the cheek) without touching or pressing on the lids/eye. If the casualty requires a head wrap, do not allow the wrap to cover the eye(s) without first shielding it, even if apparently uninjured (**d**), thereby preventing unintentional pressure on the eye. (Courtesy of Dr. Robert A. Mazzoli, MD, FACS)

wise be protected by protective eyewear should raise the suspicion of potential ocular injury and prompt shielding and further evaluation (*shield and ship*) (Fig. 13.3). Additionally, blast energies alone may be sufficient to cause significant ocular disruption even without penetration or foreign bodies, regardless of eye protection [14, 16–21].

Because initial visual acuity is an important predictor of the final visual outcome—it is the vital sign of the eye—if feasible, once the tactical situation permits, a rapid check of visual acuity should be performed and documented before protecting the eye with a shield. A useful field quantification of visual acuity is (from best to worst) (1) able to read print. While documentation through a near-vision pocket acuity card or formal Snellen chart is optimal, this is usually impractical under emergency or hazardous combat conditions. Precious time should not be lost searching for a formal acuity card at the point of injury; rather, the ability to discriminate any size or type of print—such as nametape letters or text on packaging—conveys the same information and should be attempted if possible. More formal acuity testing should be performed in a safer location once the situation permits; (2) can count the number of fingers held up (Counts Fingers, or CF); (3) can see Hand Motions (HM); (4) can see light

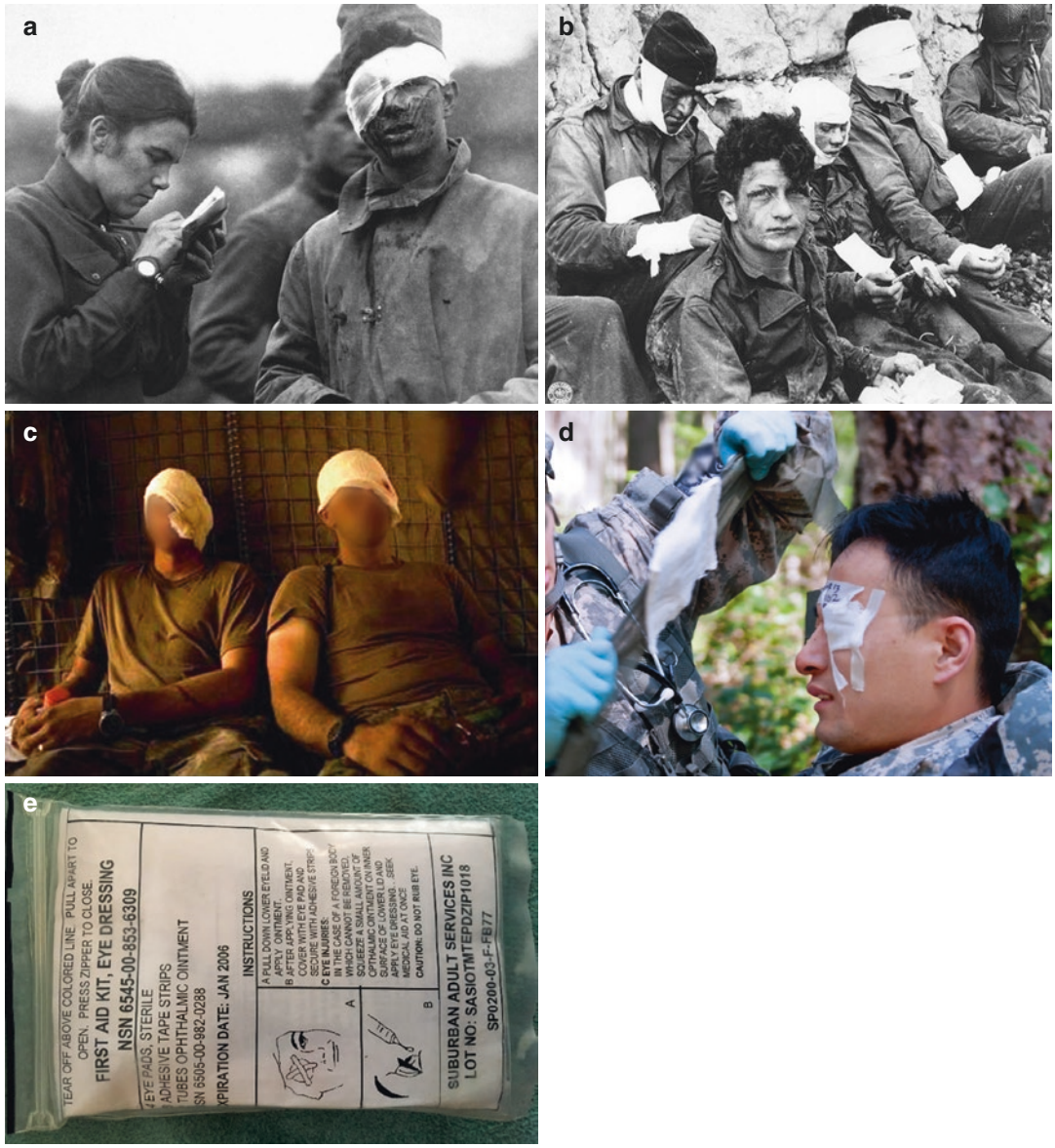

Fig. 13.2 Examples of what NOT to do for known or suspected eye injuries. Eye injuries are fundamentally different from other injuries, where direct pressure is usually the preferred initial treatment. Direct, indirect, and accidental/inadvertent pressure on an injured eye can aggravate the injury, causing herniation of delicate intraocular contents and loss of vision or the eye. Sadly, examples of patching and incorrect treatments of the eye abound and continue, including in instructional courses and equipment (**a**, from WW1. With permission World History Archive/Alamy Stock Photo; **b**, from WW2. National Archives Catalog: https://catalog.archives.gov/id/531187; **c**, from Afghanistan. Courtesy of Dr. Robert A. Mazzoli, MD, FACS; **d**, from US Army Expert Field Medical Badge (EFMB) training, 2013; **e**, An inappropriate "Eye Trauma" kit dating from the 1960s that advocates the WRONG TREATMENT of eye injuries, still included in US first aid kits until 2014. This kit has now been condemned by the Defense Logistics Agency and should be removed from wherever it is found. Courtesy of Dr. Robert A. Mazzoli, MD, FACS)

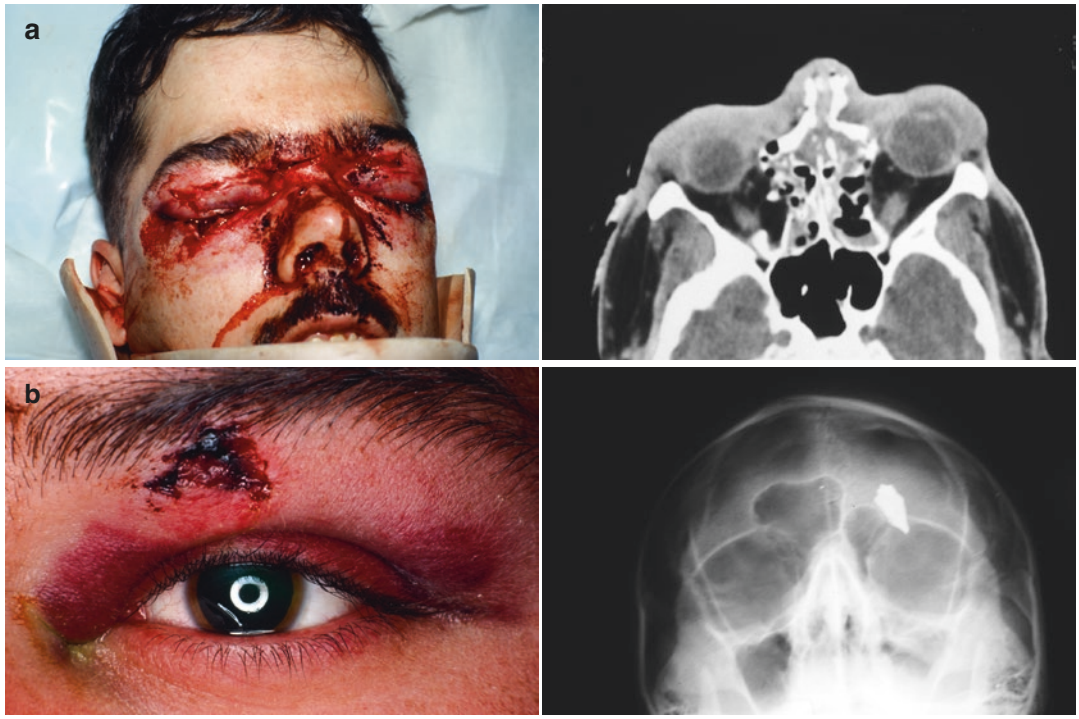

Fig. 13.3 Evaluation of eye injuries (photos from TMM, Borden Press). Have a high index of suspicion for eye injuries. Keeping in mind the mechanism of injury, grossly inspecting the face and eye area can provide initial clues as to whether the eyes are involved or not, but appearances are often deceiving. If any part of the ocular area is involved that would otherwise be protected by eye armor, presume a penetrating eye injury exists; document visual acuity, start antibiotics, and *shield and ship* the casualty for further evaluation by ophthalmology. (**a**) This patient was struck by a chain under tension which broke, striking him in the midface. Lacerations around the eyes should prompt shielding and evacuation for further evaluation. Note that while this fully conscious patient received a cervical collar at the POI because of the mechanism of injury (appropriately), he was transported without protective eye shields despite the obvious lid injuries and unknown underlying eye injuries. While he had significant naso-orbital-ethmoid fractures, amazingly, he suffered no ocular injuries. Nevertheless, not knowing that fact, the casualty should have been shielded prior to transport. (**b**) This patient was struck by a small arms blank round that was discarded into a firepit, with a portion of the casing embedded into his lid and anterior orbit. While he fortunately did not suffer a penetrating ocular injury, he suffered significant nonpenetrating corneal injury. Nevertheless, the mechanism of injury and the lid laceration indicates a high risk of penetrating injury and should prompt a shield prior to immediate evacuation from the POI. (**c**) This patient was involved in a motor vehicle crash in which the windshield shattered, peppering his face with glass shards. Although his lids and eye look relatively minimally involved, with multiple "superficial" lacerations, he had slightly decreased vision. Further evaluation revealed a penetrating ocular injury hidden by his upper lid, a consequence of an overlying full-thickness lid injury. Contrast this picture to the more dramatic photos of 3a and b, proving that appearances can be deceiving and that a high index of suspicion must be maintained. Again, the mechanism of injury coupled with injuries to the ocular area that would otherwise be protected by eye protection should trigger the placement of an eye shield and evacuation to ophthalmology; his decrease in vision elevates the evacuation to urgent. (**d**) This patient was involved in a blast. Treating medics, seeing the eyelid lacerations, placed an eye shield and evacuated the patient without examining the eye further. On closer examination, the attending ophthalmologist discovered a full-thickness scleral wound at the 12 o'clock position on the eye, hidden under the lid. Notice how relatively unimpressive the remainder of the eye appears. Because the combat medics had placed a shield and not a patch, no ocular contents were expulsed, the laceration was closed, and the eye was saved. (Courtesy of Dr. Robert A. Mazzoli, MD, FACS)

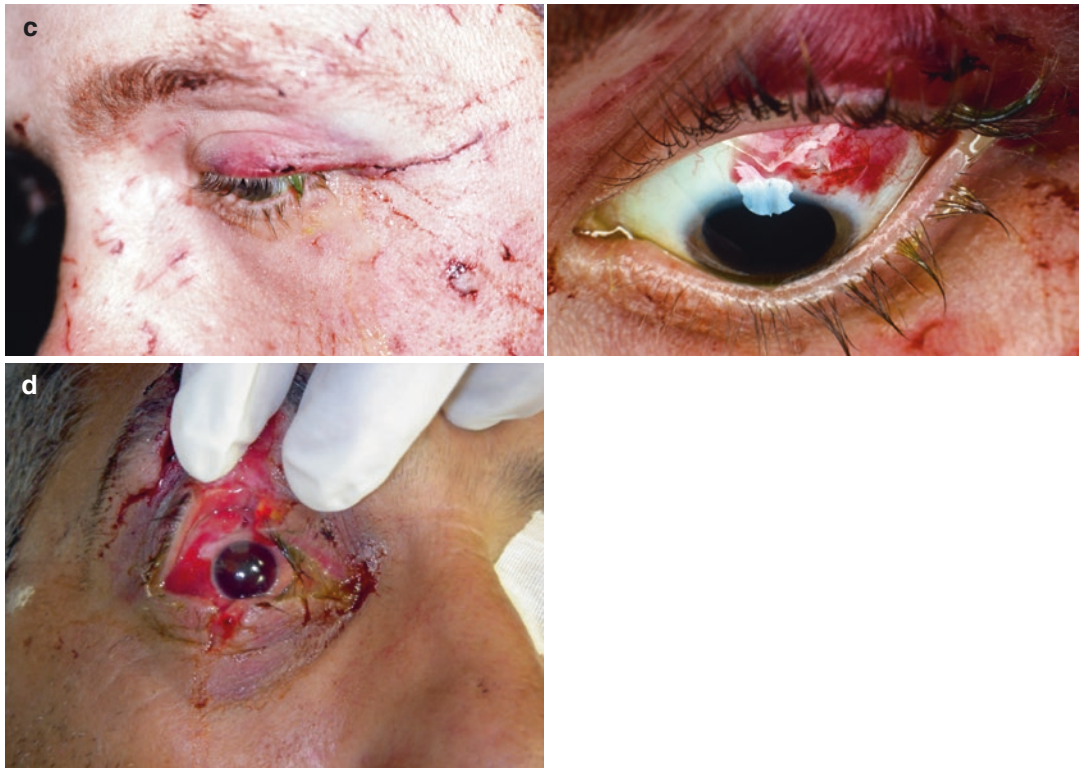

Fig. 13.3 (continued)

Table 13.2 Field testing visual acuity

Snellen chart or near-vision pocket acuity card: 20/20 through 20/400
Able to read print: specify size and distance, e.g., "Nametape @ 3 ft"
Counts fingers (CF): specify distance, e.g., CF @ 2 ft
Hand movement (HM): specify distance, e.g., HM @ 2 ft
Light perception (LP): specify distance, e.g., LP @ 2 ft
No light perception (NLP)

(Light Perception, or LP); and (5) cannot see light (No Light Perception, or NLP). Vision should be checked with the other eye closed or covered (Table 13.2). For CF, HM, or LP vision, document the furthest distance that can be discerned.

Avoid any diagnostic maneuver that places pressure on the globe. At the point of injury and in the prehospital zone, do not try to force swollen eyelids open or move them out of the way with bent paper clips or other similar devices, as suggested in some textbooks; those techniques should be performed under more controlled conditions. Do not patch the eye or place any dressings under the rigid eye shield; do not use any dressings that could put pressure on the globe; and do not place a "head wrap" over an unshielded eye (Figs. 13.1 and 13.2). Do not ultrasound the globe, even in preophthalmic facilities. Avoid maneuvers that might induce nausea, retching, and vomiting or otherwise increase central venous (intraocular) pressure. Patients suspected of having an open-globe injury (e.g., facial fragmentation injuries and lid laceration) should be treated as such, shielded, and transferred to an ophthalmologist (*shield and ship*) [22, 23].

In some cases, the eye may be penetrated, but the lacerated cornea or sclera may not be obvious. Marked eye pain or decreased vision in a combat casualty should alert the first responder to a possible open globe. Rule out a ruptured or lacerated eyeball by checking for signs such as prolapse of pigmented intraocular tissues (such as iris or lens) through a wound (Fig. 13.4). Be aware that collected debris from a blast, such as mud, dirt, and debris, may conceal herniated tissues, and that, similarly, uvea may masquerade as debris or a foreign body on the surface of the eye.

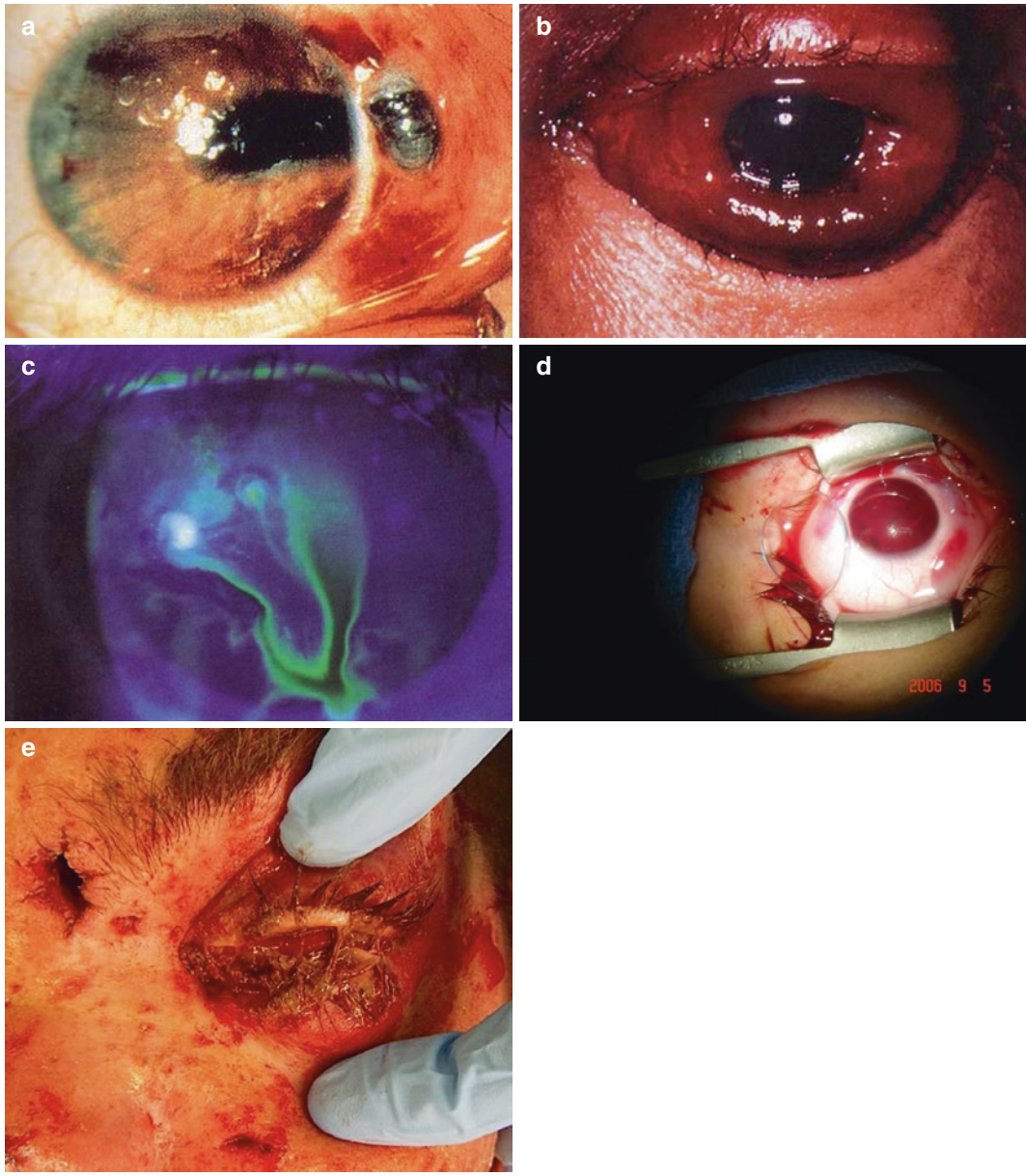

Fig. 13.4 Signs of an open globe. External signs of an open globe and serious eye injury include (**a**) prolapsed uvea and peaked pupil; (**b**) hemorrhagic swelling (chemosis) of the conjunctiva; (**c**) positive (leaking) Seidel sign, in which fluorescein stain is washed away as a rivulet by leaking aqueous humor; and (**d**) hyphema. This patient was in a blast and was not wearing eye protection. The patient was also wearing contact lenses, which were discovered at the time of evaluation. (**e**) Note that herniating ocular tissue and uvea may be disguised as or hidden by overlying debris, such as the mud in this photo of a blast injury, where discriminating tissues is essentially impossible. Debridement of this injury should be deferred to ophthalmology: *shield and ship*. (Courtesy of Dr. Robert A. Mazzoli, MD, FACS)

Debridement should therefore be approached very cautiously. Additional signs include a "peaked" pupil—that is, a distorted pupil with the "point" of the pupil pointing toward the site of the corneal or scleral laceration; hemorrhagic swelling of the conjunctiva (hemorrhagic chemosis); positive Seidel sign on the cornea; hyphema (blood in the anterior chamber); a very shallow or abnormally deep anterior chamber (compared to the uninjured eye); or severe vision loss [23, 24].

Do not apply pressure to the eye. Ask the patient not to strain or squeeze their eyelids. Tape a rigid metal Fox shield over the eye—the metal shield can be easily bent, shaped, and contoured like a moldable splint to avoid any surface contact but will still provide adequate protection—or use the casualty's own ballistic eye protection, even if slightly damaged. If a Fox shield or eyepro is not available, other alternatives include the cut out bottom of a paper cup, moldable SAM® splints, and anything rigid that can vault over the eye and orbit (Fig. 13.1) without contacting it. Do not apply a dressing or gauze patch to an open globe or under the shield. Do not use ointment on an open globe.

Prehospital Management of Combat Eye Injuries

Care for ocular injuries in the prehospital combat setting is discussed in two settings: (1) **Medic**: the far-forward combat medical provider or combat buddy operating outside of a medical treatment facility without the direct on-scene supervision of a physician or physician assistant; and (2) **Battalion Aid Station**: a Role 1 austere medical treatment facility that does not have critical care, surgical, or ophthalmic specialty expertise available, but which will typically have a physician or physician assistant. A proposed list of eye care medications and equipment is attached as Table 13.3.

Table 13.3 Battalion aid station eye care kit

Medications
Moxifloxacin 0.5% drops
Tetracaine 0.5% or proparacaine 0.5%
Prednisolone acetate 1% drops
Moxifloxacin 400 mg tabs
Levofloxacin 500 mg tabs
Bacitracin ophthalmic or erythromycin ophthalmic ointment
Prednisone 20 mg tabs
Artificial tears
Scopolamine 0.25% drops
Diclofenac 0.1% drops
Pilocarpine 2% drops
Miscellaneous
Penlight with blue filter
Fluorescein strips
Cotton-tipped applicators
Metal or rigid plastic eye shield
Tape (1 inch, plastic or nylon)
Near-vision card
Wound closure strips (1/4 inch)
Magnifying glass
Fine forceps

Adapted from Butler [24]

Penetrating or Suspected Penetrating Eye Trauma

Medic

When the eye sustains a direct hit from a military assault rifle, preservation of vision is unlikely, and preservation of life is often the primary concern. Explosions, however, which may include artillery or mortar shells, shoulder-fired rocket shells, and improvised explosive devices, create multitudes of small, high-velocity fragments. This low-mass, high-energy shrapnel may cause relatively innocuous injuries elsewhere but can devastate the eye. Nevertheless, every attempt to protect the eye from further trauma should be made, since such ocular injuries do not preclude the possibility of salvaging useful vision in the eye with timely and proper surgical care.

If the operational situation allows, perform a rapid assessment of visual acuity as outlined above. If the lids are too swollen for the casualty to open them, do not attempt to pry them open; assume a significant eye injury, deferring visual acuity to a higher echelon. Administer oral moxifloxacin from the Combat Wound Medication Pack, place a rigid eye shield, and evacuate the casualty to a higher echelon (*shield and ship*).

Evacuation priority should ensure the casualty arrives at an ophthalmic facility as soon as possible but no later than 12 hours (Urgent or equivalent).

When penetrating eye trauma is obvious or is suspected, the most important aspect of first responder care is to protect the eye with a rigid shield to prevent any inadvertent pressure or additional trauma that could result in the expul-

sion of intraocular contents through the corneal or scleral defect. An effective eye shield vaults the eye rigidly without touching it, preferentially resting on the bones of the brow and cheek; anything that meets that requirement can act as an effective eye shield [22–27].

Of particular note is the need to avoid placing any dressing or bandage that puts pressure on a potentially open globe. Unlike hemorrhagic injuries elsewhere, pressure dressings and patches are not part of the care of an eye injured in combat and may result in avoidable and permanent loss of vision [22–27]. Mazzoli documented that this seemingly obvious aspect of care for the combat casualty was often not performed or was performed incorrectly [28]. The presence of incorrect guidance on optimal management in several Army doctrinal documents as well as the presence in the DoD supply system of antiquated and ill-advised eye first-aid kits that included pressure dressings was documented by Sauer in 2014 and may help account for the unexpectedly large failure to comply with this aspect of combat casualty care (Fig. 13.2) [29]. Other factors contributing to poor compliance include the chaos of combat, the co-existence of other head and facial wounds that may be more dramatic, the disguising of ocular injuries with mud, dirt, blood, and other debris, and the simple availability of shields [28]. Brunstetter noted that rigid eye shields are not a component of most service individual first-aid kits, but this situation is being rectified; commercially manufactured shields are increasingly found in point-of-injury aid kits, such as the Individual and Joint First Aid Kits (IFAK-II, JFAK), the Combat Medic's Aid Bag, and the vehicular Warrior Aid and Litter Kit (WALK) [30–32]. Additionally, although the JFAK (which does include eye shields) will be adopted as the joint-service standard for point-of-injury equipment, [32] there may still be significant differences between equipment issued in garrison and for deployment [30–32]. The need to avoid pressure dressings and to use rigid eye shields for the management of known or suspected penetrating eye trauma was specifically addressed by Assistant Secretary of Defense for Health Affairs Jonathan Woodson in his memo on optimal management of combat eye injuries dated 7 July 2014 [27]. and by the Commander of U.S. Forces, Afghanistan, in 2013 [25].

If no eye shield is available, use the casualty's own ballistic eye armor, even if slightly damaged. This will also help prevent added debris from helicopter rotorwash from entering the eye(s) during evacuation. Alternately, use any other field-expedient device that does not place pressure on the globe and which vaults cleanly over the underlying eye. Examples of other field expedients include moldable splint material (SAM® splint) or the bottom of a drinking cup or plastic bottle. An eye shield, BY DEFINITION, does not contact the eye and has no dressing underneath it.

The shield should remain on the eye at every echelon of nonophthalmic care. If the eyelids cannot be opened because of swelling or bruising, suspect an open globe and transfer to an ophthalmologist. DO NOT attempt to forcibly open the lids at this point of care. *shield and ship* [23].

Battalion Aid Station

As per medic, obtain a more accurate check of visual acuity at the first opportunity (e.g., Snellen or near card testing). Start or continue systemic antibiotics, preferably intravenous fourth-generation fluoroquinolone. Administer antinausea and anti-emetic (e.g., ondansetron (Zofran®)), analgesics, and tetanus (if needed). Analgesic doses of ketamine are not contraindicated in ocular injuries [22, 23]. Ensure a rigid eye shield is in place without an underlying eye patch prior to evacuation. *shield and ship* to ophthalmic care as soon as possible (Urgent) so that any open globe can be addressed within 12 hours.

While some authors have argued that more advanced ocular diagnostic techniques such as ultrasound can be used by nonophthalmologists to help detect the presence of corneo-scleral lacerations or intraocular foreign bodies, [33, 34] this is not recommended. Beyond direct observation, the standard and preferred primary imaging modality for ocular trauma is thin-slice axial CT with coronal reconstruction, which

Table 13.4 Tactical combat casualty care (TC3) guidelines for ocular injuries

Perform a rapid field test of visual acuity
Cover the eye with a rigid eye shield (NOT a pressure patch)
Administer appropriate systemic antibiotics

provides exceptional detail of both soft tissue and bony structures. Understandably, availability of CT is clearly limited in an operational environment, especially at this echelon of care. Ultrasound, though, even in controlled stateside environments, has the potential for applying pressure to an open globe and aggravating the injury in other ways and would be much riskier in a combat location where immediate ophthalmic intervention is not available. This risk increases further in the hands of the occasional ocular ultrasonographer, especially in the midst of medical emergencies. More practically, ocular ultrasound adds little information that would appreciably alter the management of a suspected eye injury at this echelon, that is, *shield and ship* [35–39]. MRI is contraindicated because of the potential of retained magnetic foreign body. Checking the vision, shielding the potentially penetrated eye, urgent evacuation, and preventing intraocular infection through the administration of a systemic fourth-generation fluoroquinolone that has good penetrance into the vitreous space [40–42] are the mainstays of Tactical Combat Casualty Care (TC3) management (Table 13.4) [22, 38, 39].

Post-Traumatic Endophthalmitis

Post-traumatic endophthalmitis is an infection of the anterior and posterior chambers inside the eye. This type of intraocular infection typically has devastating visual results. Risk factors include presence of intraocular foreign body (IOFB); lens rupture; delayed primary globe repair longer than 24 hours; rural trauma; trauma with contaminated objects; vitreous prolapse; and afferent pupillary defect [43, 44]. Final visual outcome is highly variable, with development of retinal detachment, virulence of the organisms, location and size of the wound, and nature of the IOFB all affecting final visual acuity. Gram-positive cocci were the most common organisms found in one study of 347 cases of post-traumatic endophthalmitis, followed by *Bacillus subtilis*, *Pseudomonas aeruginosa*, and *Escherichia coli* [45]. *Bacillus cereus* is another very aggressive pathogen often isolated in intraocular infections, particularly from rural areas or soil-contaminated injuries, and is associated with dramatically rapid deterioration and poor visual outcomes, notoriously progressing to significant pain and total blindness within 18–24 hours. As testament to its virulence, while 50% of eyes infected with the relatively low-virulence *Staphylococcus epidermidis* achieved a final visual acuity of 20/400 or better, a majority of eyes infected with gram-negative organisms or *Bacillus* commonly result in total loss of vision and enucleation [43]. Of note, delayed removal of intraocular foreign bodies at CONUS facilities for 79 eyes injured during Operations Iraqi Freedom and Enduring Freedom was found to result in no cases of endophthalmitis if systemic antibiotic therapy was maintained during evacuation and topical antibiotic drops were added after primary closure of the corneal or scleral defect by an ophthalmologist [46]. Consequently, a casualty with penetrating eye trauma needs broad-spectrum coverage by an antibiotic that has good intraocular vitreous penetration, such as systemic third- or fourth-generation fluoroquinolones.

Medic

Moxifloxacin, 400 mg once a day (as found in the Combat Wound Medication Pack) given as soon as possible after wounding and continued until arrival at ophthalmic care, is the treatment of choice in the prehospital environment. Other antibiotics may not penetrate the vitreous well and should not be substituted for moxifloxacin without consulting with an ophthalmologist. The casualty should be evacuated as soon as feasible (Urgent), as poorer visual outcomes are associated with increasing delays in wound closure and treatment beyond 12–24 hours. Do not use

antibiotic eye drops or ointments in an eye with penetrating eye trauma until after the casualty has reached ophthalmic care.

The recommendations above are outlined in the JTS CPG and the Tactical Combat Care Guidelines (TC3 Guidelines) and are consistent with the recommendations in the Emergency War Surgery Manual and with the recommendations for managing eye trauma in austere environments found in Auerbach's textbook, Wilderness Medicine [22–24, 39–42].

Battalion Aid Station

Same as for medic except that at this point, IV antibiotics may be started. Use fluoroquinolone antibiotics such as moxifloxacin or levofloxacin and begin an anti-emetic as needed to prevent vomiting (ondansetron (Zofran®) 4 mg IV). Avoid interventions that may induce nausea/vomiting. Do not administer ointments or drops. Do not perform ocular ultrasound. Do not patch the eye or place wet gauze over it, as the "wet-to-dry" effect may debride intraocular contents on removal. Give tetanus prophylaxis. *shield and ship*. Evacuate expeditiously (Urgent) to ophthalmic care to ensure that evaluation and globe surgery can be accomplished within 12 hours of injury.

Eyelid Lacerations

Eyelid lacerations are commonly seen in combat and may or may not be accompanied by penetrating eye injury. Be aware that visible fat herniating through a lid laceration is, by definition, a sign of deeper, more significant orbital injury and requires specialized evaluation and care by an ophthalmologist. Do not debride or excise lid tissue. When evaluating a lid laceration, be mindful that the skin of the lid is the thinnest of the body (less than 1 mm), with other important lid structures residing within millimeters of the surface, and that what may seem to be a "simple" or "superficial" injury may actually be quite significant, even if only 5 mm deep.

Medic

Do not attempt to repair eyelid lacerations in the prehospital environment. Give the casualty his or her Combat Wound Medication Pack—then *shield and ship*.

Battalion Aid Station

Minor and linear lid lacerations that do not involve the lid margin or tarsus, the orbit, or the medial or lateral canthus may be repaired at the BAS level using inert sutures such as 6-0 nylon or polypropylene sutures (on small needles such as Ethicon®-equivalent P-3), as long as there is no suspicion of penetrating injury to the eye or need for more expert attention. More complex lid lacerations should be *shielded* and *shipped* without wet gauze applied. Inspect medial lacerations carefully for lacrimal canalicular injury, including gentle lateral traction, as these lacerations can spontaneously reapproximate and mask the true extent of the injury. If in doubt, *shield and ship*. In repairing "simple" lid lacerations, beware of passing sutures more deeply than 1–2 mm so as to not incarcerate other tissues, which can lead to cicatricial lid retraction and corneal exposure. Deep sutures and layered closure are not usually indicated or recommended. Avoid braided or absorbable sutures in ocular and lid trauma [i.e., silk, polyglactin/polyglycolic acid/polydioxanone (Vicryl®/Dexon®/PDS®)]. Apply topical ophthalmic antibiotic ointment. Remove sutures in 5–7 days.

Eye Injuries from Blast

Most recent combat eye injuries are the result of blast and blast effects of high-energy fragmentary munitions rather than gunshot wounds [3, 4, 15, 47]. Injuries may occur in casualties exposed to blast forces despite the use of protective eyewear [4, 14, 48–50]. Upward-directed blast forces may displace the protective eye wear and allow penetrating fragment injury to the ocular surface and orbit. Eye injuries are a common

finding in blast polytrauma: over 30% of head injuries will have associated eye injuries [6, 9, 10]. The size, type, distance, and location of the explosive charge as well as the amount and type of materials that may generate fragments from the blast may all affect the resulting injury patterns [6]. Additionally, the blast wave itself may cause closed-globe injuries to the retina or other intraocular structures [12, 14, 16–21, 50, 51]. Be aware that "simple" corneal abrasions or corneal foreign bodies in the setting of a high-energy blast debris wind that has gotten past eye protection may be an indicator of more severe concussive intraocular injury [13, 14]. The blast may also cause post-traumatic inflammation of the eye, which may result in eye pain and decreased vision. Casualties who describe vision loss or eye pain after exposure to blast should have their vision documented and also be treated with a protective shield, but antibiotics are not indicated unless there is suspicion of penetrating eye injury. Visual dysfunction such as postblast light sensitivity (photophobia), difficulty with reading and near vision, and diplopia may also accompany blast-related traumatic brain injury (TBI) and should be evaluated by ocular specialists [52–55].

Medic

Have a high index of suspicion for penetrating and blunt eye injuries after a blast. Check and document visual acuity if the casualty's condition and operational situation allow. If any portion of the eye and adnexa that would otherwise be protected by eye protection is injured—including "simple" corneal abrasions—*shield and ship*, apply a rigid eye shield without an underlying patch or gauze and evacuate to ophthalmology. If treating concomitant head or facial injuries, do not allow the head wrap or dressing to cover eyes without first shielding the eye(s) with a rigid eye shield, even if the eyes appear uninjured. Do not apply ointments or drops into the eye. If the lids are too swollen to allow examination, do not manipulate them or try to pry them open; simply *shield and ship*.

Battalion Aid Station

As for medic. Document visual acuity and confrontation visual field in each eye separately. Treat superficial injuries such as corneal abrasions but be aware that corneal abrasions and foreign bodies may be an indicator of more serious intraocular injury. Examine the eyes and orbits thoroughly but gently, including fluorescein staining. Consider the possibility of penetrating globe injury and expulsion of intraocular contents prior to debriding or irrigating the eye, as uveal tissue and intraocular contents can look very similar to debris. When irrigating eyes, use only sterile eye wash or normal saline; do not use water. If the lids are too swollen to allow examination, do not manipulate them or try to pry them open and do not perform ocular ultrasound. If the orbit is extremely tense and proptotic from retrobulbar hemorrhage, perform emergency canthotomy and cantholysis. Shield and transfer for ophthalmic evaluation if vision is decreased or deteriorates.

Blunt Trauma to the Globe

Motor vehicle accidents are a common cause of blunt force injuries, including eye trauma, in combat forces [15, 56]. Other possible causes of blunt trauma include falls, noncombat sports or other activities, operational accidents (i.e., from bungee cords, radio antennas, vehicle springs, tie-down straps, etc.), and primary, secondary, and tertiary blast injury effects (Table 13.5) [6, 9, 11, 22, 56]. Common anterior injuries include superficial conjunctival and corneal injuries but can include significant injuries such as hyphema, traumatic iritis, traumatic iris or ciliary body disinsertion, and pupillary dysfunction (pupillary sphincter tears and traumatic mydriasis) [12]. Posterior segment injuries can also be significant and vision threatening, including lens dislocation, vitreous hemorrhage, retinal contusion and photoreceptor damage (commotio retinae), retinal detachment, choroidal detachment or rupture, optic nerve avulsion, traumatic optic neuropathy, and frank globe rupture [57]. While antibiotics

Table 13.5 Blast-related nonpenetrating blunt ocular injuries

Corneal abrasion
Corneal foreign bodies
Corneal/endothelial cell contusion
Corneal edema
Hyphema
Traumatic iritis
Anterior chamber angle recession
Iridodialysis
Pupillary/iris sphincter tear
Traumatic mydriasis
Zonular disruption/lens dislocation
Cyclodialysis
Vitreous hemorrhage
Choroidal hemorrhage
Choroidal rupture
Choroidal detachment
Commotio retinae/photoreceptor damage/Berlin's edema
Retinal detachment
Macular hole
Traumatic optic neuropathy
Optic nerve avulsion
Scleral/globe rupture
Orbital hemorrhage
Orbital compartment syndrome
Orbital fracture

are not needed or indicated for closed-globe injuries, if there is any doubt about ocular integrity or an open globe, prudence dictates antibiotics be started until an ophthalmologist can more fully evaluate it.

Medic

Casualties injured by blunt force mechanisms who report vision loss or eye pain should also be treated with a protective shield. Because the eye can appear normal externally, a high index of suspicion for significant intraocular injury must be maintained. The eye is otherwise largely treated as if it were a penetrating ocular injury—check visual acuity and *shield and ship*—but antibiotics are not indicated IF an open globe can be definitively ruled out on exam (**Caveat**: do not perform ocular ultrasound to determine this). If there is suspicion of penetrating eye injury (i.e., any overlying lid laceration), the management strategies outlined above for penetrating eye injury should be followed; administer the Combat Wound Medication Pack antibiotic until a more thorough examination can be performed. Apply a rigid eye shield and evacuate to ophthalmology to arrive within 12 hours. Elevating the head will allow intraocular blood to settle with gravity and may help clear the vision temporarily. Take measures to prevent nausea, retching, or vomiting.

Battalion Aid Station

As per medic. Document vision more accurately. Examine the eye more thoroughly with direct observation to the best of one's ability. Again, do not perform ocular ultrasound at this echelon. Check for orbital compartment syndrome as described below. If vision is compromised, *shield and ship* to ophthalmic care for evaluation.

Laser Eye Injury

Although blinding lasers are forbidden by international laws of combat and there are not yet any weaponized offensive blinding laser weapons known to be fielded [58], lasers are in common use on the battlefield for purposes such as range-finding, nonlethal escalation of force (EOF), and target designation. The military uses both visible-light lasers ("green beam" lasers, e.g., EOF designators, laser dazzlers, etc.) and invisible lasers (e.g., target designation). While military devices are recognized as powerful enough to pose ocular hazards, increasingly powerful handheld lasers are becoming widely available via the internet and may find their way to the barracks and battlefield. In most instances, ocular exposure to visible-light lasers results only in temporary "photoflash blindness" with no permanent ocular damage or injury, a condition known as an "illumination" or a "bright light event" (i.e., dazzle). Of important note, this *does not* equal a true "laser injury." In fact, the risk of a true "injury" from a visible-light laser is extremely small (but the risk is definitely real). However, even

the temporary dazzling effect can dramatically affect operational capability, such as when dazzled while driving or flying an aircraft, particularly in darkened conditions. Indeed, the Federal Aviation Administration (FAA) reports a 14-fold increase in the number of civil aviation-related laser incidents from 2006 to 2014, with almost 4000 cockpit illuminations reported in 2013; while a federal offense and still posing a significant risk to aviation safety, no permanent injuries were reported [59–62]. On the other hand, invisible lasers, by their energies and mechanisms of action, can be more dramatically injurious with nearly instantaneous loss of vision and permanent ocular damage [63]. Nevertheless, both types of laser can be hazardous and pose true ocular risk, particularly if the eye is exposed within the Nominal Ocular Hazard Distance (NOHD), a safety distance specified for each particular laser [64]. With either type of laser, optical magnification (i.e., binoculars and sighting systems) can increase the ocular hazard. Most laser injuries are accidental, being either unintentionally self-inflicted, the result of horseplay (i.e., "laser tag" or "light saber" battles) or the result of friendly illumination but can still be the result of enemy action; Mader et al. reported one such injury from Operation Desert Storm [15]. While laser-protective lenses are available, to be effective the light absorption spectrum of the lens must be specific to the wavelength of the laser being used; if various wavelengths are fielded, separate lenses may need to be used for each type of laser identified as a threat, or alternatively, the protective lens chosen for use should be protective against the most significant known laser threat. Additionally, the lens coloration degrades operational vision much as sunglasses do. Consequently, their use in nighttime operations should be weighed against operational requirements [63, 64]. Because of the psychological impact and the operational toll laser exposures and injuries can carry, it is incumbent on leaders to ensure proper training with all types of lasers, essentially treating them as loaded weapons [64].

Symptoms of laser eye exposure may vary with the type of laser involved. Visible-light lasers (dazzlers and EOF designators) will typically cause a temporary bright light glare and dazzle, as in a photoflash. An afterimage may appear and persist but should resolve on its own. Nonetheless, this can significantly degrade operational effectiveness in the meantime, leading to secondary injuries. Symptoms may linger longer in nighttime exposures. Be aware that even visible-light lasers can cause significant true ocular damage if operated within the NOHD.

Invisible lasers typically cause sudden painless loss or distortion of vision, commonly causing intraocular hemorrhage. The loss of vision may be profound and persistent, either as loss of central visual acuity, distortion of objects, and straight lines (metamorphopsia) or as persistent blind spots. The casualty may be aware of seeing a flash of light just prior to the vision loss. Light flashes may not be noticed if the causative laser beam is in the infrared portion of the light spectrum and therefore not visible to the human eye. Laser damage to the retina should be suspected in any patient with visual loss on the battlefield who has no obvious signs of external injury [58, 63, 64].

Visible-light laser exposures generally require no treatment if visual symptoms abate. True laser injuries primarily affect the retinal photoreceptors. While there are few prehospital interventions that effectively treat posterior segment laser injuries, [58, 63, 65] high-dose oral steroids, and non-steroidals (indomethacin) have been shown to improve photoreceptor survival [66]. A recent case report anecdotally reported good recovery of vision after treating a dense sub-foveal hemorrhage with intravitreal ranibizumab, a VEGF inhibitor, [67] but this would not be a treatment for preophthalmic echelons of care. The vision loss caused by laser-induced retinal damage often improves significantly over time [65, 68].

The most important aspect of managing combat laser eye injuries is to implement preventive countermeasures to reinforce laser safety and to prevent further laser injury both to the casualty and to the other unit members. Unit training should include laser safety briefings on hazards, control measures, illumination of friendly forces, and proper employment of the devices, particu-

larly within the NOHD [64]. Members of the unit should be warned to avoid looking directly at any sources of bright lights. Use caution if optical sighting devices such as binoculars and rangefinders are being used, since these devices may magnify laser injury to the eye. If available and if operational requirements allow, laser eye protection should be used; this type of eye protection, however, must be specific to the light wavelength of the threat laser to be effective and may degrade operational performance. Other measures such as taking cover or setting up a smoke screen can be useful. Indirect viewing methods for target acquisition and surveillance may be employed as well. Friendly laser sources should be used with caution; they should not be aimed at other unit personnel, and users should avoid looking at the output end [63, 64].

Medic

If presented with a potential laser injury or illumination, immediately alert other unit personnel of the laser threat. Document visual acuity in each eye separately. Ask specifically about distortion of vision (metamorphopsia) and blind spots (scotomas). Gather data surrounding the date, time, type of laser (visible vs. invisible, and model, if known), and circumstances of the potential exposure (distance, duration of exposure, optics used, daytime vs. nighttime, etc.). If vision is normal, the person may return to duty. There is no need for eye drops or ointments. If acuity is decreased or visual symptoms persist, refer to higher echelon for further evaluation.

Battalion Aid Station

As for medic. Document visual acuity in each eye separately. Examine each eye for metamorphopsia with an Amsler grid (available online) (Fig. 13.5), a piece of graph paper, or by asking if straight lines appear wavy. Funduscopy may reveal intraocular or retinal hemorrhage. If examination is normal, may return to duty; no drops or ointments are necessary. If acuity is decreased or metamorphopsia is present, begin oral NSAID such as indomethacin 25 mg po TID or equivalent. Suspected laser injuries should be referred promptly to ophthalmology/optometry and the incident reported up the chain of command. Specifically, a report should be made to the theater Laser Safety Officer and the DoD Laser Injury Hotline, at https://hpws.afrl.af.mil/dhp/OE/ESOHSC/laserinjury [69].

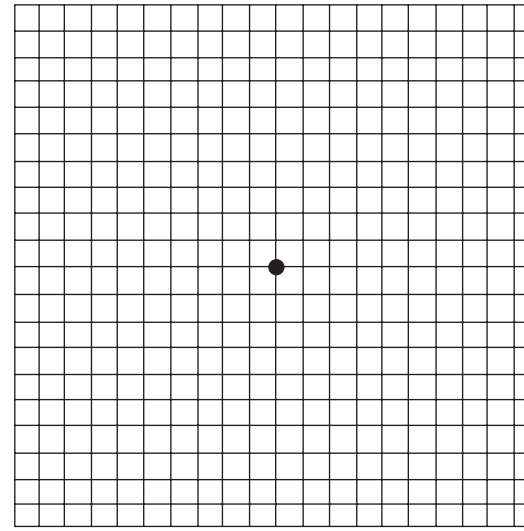

Fig. 13.5 Amsler grid. An Amsler grid can reveal areas of distorted vision (metamorphopsia), an important finding in potential laser injuries and other conditions affecting the macular area of the retina. Test each eye separately, with glasses or correction (start with the unaffected eye if symptoms are unilateral). Have the patient look at the center spot. While looking at the center spot, the remainder of the grid should still be visible. The lines should be perfectly straight, both vertically and horizontally. There should be no distortion or waviness of the lines, and there should be no blind spots. Have the patient draw in areas of distortion (metamorphopsia) or scotoma. Lacking a grid, any graph paper can substitute, as can asking about straight lines in nature (e.g., door jambs), but an Amsler grid is standardized. (Courtesy of Dr. Robert A. Mazzoli, MD, FACS)

Chemical Injury

Serious injury to the eye from chemical weapons on the battlefield is uncommon, although an increasing number of nations are reported to be acquiring, developing, stockpiling—and now

using—such munitions. Nevertheless, chemical injury in deployed and training environments from other chemical agents is not uncommon, given the wide variety of products used by the military. Common culprits include industrial detergents used in shipboard, aircraft, and motor-pool maintenance activities, solvents and cleaning compounds, chemical training or crowd control agents, and even common laundry detergent and household cleaners available in more developed deployed environments. Understandably, industrial strength agents are more concerning than commercial-grade agents; however, even home-use products can cause considerable damage. The chemicals most injurious to the ocular surface are strong acids (such as car battery acids or cleaning acids) and alkalis (such as bleach, detergents, or plumbing products), with alkalis posing the greatest danger. Other common chemicals such as petroleum products may irritate the eye and result in transient loss of vision but typically do not cause sustained damage to the cornea or ocular structures.

Medic

The mainstay of treating chemical eye injuries is to flush the ocular surface to remove the chemical agent. IMMEDIATELY BEGIN COPIOUS IRRIGATION. The preferred fluids are lactated Ringers, normal saline, or Plasma-Lyte A IV fluid if available. If these fluids are not available, use potable water. If none of these options are available, use the cleanest water immediately available. Do NOT try to neutralize acidic or basic chemicals that may have injured the eye. Just flush the eye as noted above [23, 24]. Irrigate for 60 minutes if feasible, then *shield and ship*.

Battalion Aid Station

As for medic. Continue copious irrigation. Examine the eyes for signs of ocular ischemia such as corneal clouding and avascularity of the conjunctiva and sclera, especially if the culprit agent is an alkali. Use topical anesthetic liberally while irrigating to ease the irritation of irrigation. Check ocular pH with urine dipsticks if available; continue irrigation until neutrality is reached (compared to the uninvolved eye). Evert the lids and examine for retained particulate matter. Sweep the upper and lower conjunctival fornices with a moistened cotton-tipped applicator to remove particles. After irrigation, document vision (which may be slightly reduced secondary to a temporary keratopathy from irrigation). Treat with topical ophthalmic antibiotic ointment. If signs of ischemia, contact ophthalmology, shield the eye, and evacuate immediately while continuing irrigation.

Conjunctival Foreign Bodies

Medic

If a penetrating injury to the globe is obvious or suspected based on physical findings or the mechanism of injury (i.e., unprotected exposure to a blast, grinding, etc.), do not attempt to remove foreign particles from the eye. *shield and ship* and treat as per penetrating eye injury, above.

If a superficial foreign body is seen or suspected and there is no significant potential for a penetrating globe injury, use lactated Ringers, normal saline, or Plasma-Lyte A IV fluid to attempt to irrigate the foreign material out of the eye. If these fluids are not available, potable water may be used. DO NOT remove impaled or stubborn foreign bodies from either the lids, the orbits, or the eyes. *shield and ship*.

Battalion Aid Station

As per medic, but in this setting, as long as there is no suspicion of penetrating ocular injury, the eye may be anesthetized with topical tetracaine and the foreign body may be removed with a Q-tip or fine forceps with the aid of light and magnification. Treat with topical ophthalmic

ointment but do not patch. Do not dispense topical anesthetic as a treatment. Evacuate persistent or deep foreign bodies that are resistant to gentle removal; *shield and ship* to ophthalmology/optometry.

Corneal Abrasions

Corneal abrasions on the battlefield should be diagnosed with caution. The term as used here means a traumatic disruption of the protective external layer of cells on the cornea (the epithelium). The traumatic event is typically immediately obvious (a fingernail or tree branch brushed across the surface of an unprotected eye). The onset of eye pain and tearing is immediate, often accompanied by a decrease in vision. DO NOT make the diagnosis of a corneal abrasion if the physical findings or mechanism of injury suggest the possibility of penetrating injury to the eye or orbit. Be particularly suspicious if the mechanism involves any kind of blast or hammering/grinding/sandblasting metal-on-metal.

Likewise, be mindful of contact lens-associated problems (even though contact lens wear is not authorized in field or deployment environments), as these problems may quickly become vision-threatening infectious corneal ulcers. Understanding that laser refractive surgery is increasingly common to enhance operational performance, particularly LASIK, be aware that a LASIK flap may rarely become dislocated by ocular trauma and mimic a displaced contact lens. Do not attempt to remove it; simply *shield and ship*.

Medic

Casualties with moderate-to-severe corneal abrasions will typically not be able to function as combatants until the abrasion is healed. The eye should be covered with a shield and the casualty evacuated to a Battalion Aid Station if feasible. Do not patch.

Battalion Aid Station

As for medic. Ensure there is no penetrating eye injury. Ask about and inspect for contact lens wear but ask about prior laser refractive surgery as well (particularly LASIK). Document vision in each eye separately. Verify corneal abrasion via fluorescein staining. Treat corneal abrasions with ophthalmic antibiotic drops (such as moxifloxacin or gatifloxacin) or antibiotic ointment. Do not use topical steroid drops. Do not dispense topical anesthetics as a treatment; treat pain with oral pain medications if necessary. Follow the patient daily until the epithelium heals and vision returns to normal. If there are any corneal infiltrates, opacities, or ulcers, especially if there is a history of contact lens wear, or if there is a potential of a dislocated LASIK flap, evacuate immediately to ophthalmology/optometry.

Orbital Compartment Syndrome

Penetrating and blunt injuries to the orbit may cause bleeding in the retrobulbar space (retrobulbar hemorrhage). Because the bony orbit is rigid, the only direction for decompression is anteriorly but this is limited by the restraining action of the lids and medial and lateral canthal tendons, which may cause increased orbital and intraocular pressure, creating an orbital compartment syndrome (OCS). This results in pain, proptosis (forward bulging of the eye), and decreased ocular movement and may cause damage to the optic nerve and permanent loss of vision [70]. Other potential causes of orbital compartment syndrome include, among others, subperiosteal abscess or hemorrhage, overaggressive fluid resuscitation (i.e., burn resuscitation) and expanding air in an orbit after orbital fracture (pneumoorbita). Pneumoorbita and subperiosteal hemorrhage have occurred in divers on ascent (without vision loss) and, theoretically, could occur while transporting a casualty in hypobaric conditions (i.e., during air transport as gasses expand) or after sneezing forcefully after trauma [70–73]. OCS is a true ocular emergency. Treatment is emergent lateral

canthotomy and cantholysis of the lower lid and other maneuvers to decrease the intraorbital pressure. Because permanent loss of vision can result within about 60–90 minutes, treatment in a combat zone cannot afford the delay that evacuation to an ophthalmologist requires. Skilled and trained providers must be able to perform this vision-saving procedure prior to evacuation.

Orbital compartment syndrome should be suspected if there is increasing pain and protrusion of the eye from the orbit after trauma, although proptosis may be hard to appreciate due to lid edema. There typically is also decreasing vision and ocular motility [70]. The orbit may be "rock hard" to gentle palpation. Orbital hemorrhage and OCS may occur at the time of the injury or may be delayed in onset; consequently, check vision frequently in orbital injuries, particularly during evacuation.

Medic

It is difficult to determine with certainty that there is no penetrating injury to the globe in most battlefield settings, so casualties with suspected orbital compartment syndrome should be managed with a rigid eye shield, oral antibiotics, and emergent evacuation to the closest next echelon. Instruct casualties with suspected orbital fractures not to blow their nose or to stifle sneezes (sneeze with mouth open) to minimize increased Valsalva/central venous pressure that can create pneumoorbita. Combat medics should not attempt lateral canthotomy/cantholysis.

Battalion Aid Station

If orbital compartment syndrome is suspected, there is not a strong suspicion of penetrating eye injury, and there is an individual who has been trained to perform the procedure, an emergency lateral canthotomy and cantholysis, a relatively simple and vision-saving procedure, should be performed as soon as feasible, as permanent damage can develop quickly and cannot be delayed for evacuation to the ophthalmologist. Small, blunt-tipped scissors are essential; because of the risk of injuring the globe, do not use sharp-pointed scissors for this procedure (Fig. 13.6). This will relieve the compartment syndrome by allowing the eye to move forward and lowering the intraocular and intraorbital pressures. The casualty should still be evacuated emergently to a facility where an ophthalmologist is available [70–74]. Do not attempt needle decompression of tension pneumoorbita or subperiosteal processes. For pneumoorbita at altitude, increase cabin pressure or descend. If the condition does not resolve promptly, perform canthotomy/cantholysis.

Ballistic Eye Protection

The most important aspect of ocular injuries on the battlefield is prevention. Properly worn ballistic eye protection made of military-grade polycarbonate plastic is essential for preserving both combatant vision and the fighting force. Ballistic eye protection has been documented to reduce both the incidence and the severity of eye injuries in combat casualties [3–5]. The importance of eye protection was emphasized in a 2008 All Army Activities message (ALARACT) that noted that only 5% of 261 casualties were known to have been wearing eye protection and that significant eye injuries were present in 8.2% of all injured warriors admitted to a Role 3 medical treatment facility [26]. A review of 3276 casualties in Iraq found that 26% of those who did not wear ballistic eye protection suffered eye injuries, whereas only 17% of those documented to have been wearing eye protection suffered eye injuries ($p < 0.01$) [4]. Breeze estimated that eye injuries could be reduced from the 4.5% incidence observed in his study cohort of UK combat casualties to 0.5% if existing eye protection had been worn [75]. In his report of a single unit's deployment experience, Gondusky reported exactly this rate of eye injuries (0.5%) despite 53% head and neck injuries suffered during the collection period and directly attributes the low rate of eye casualties to near-100% compliance with wearing eye protection [3]. The Commander of

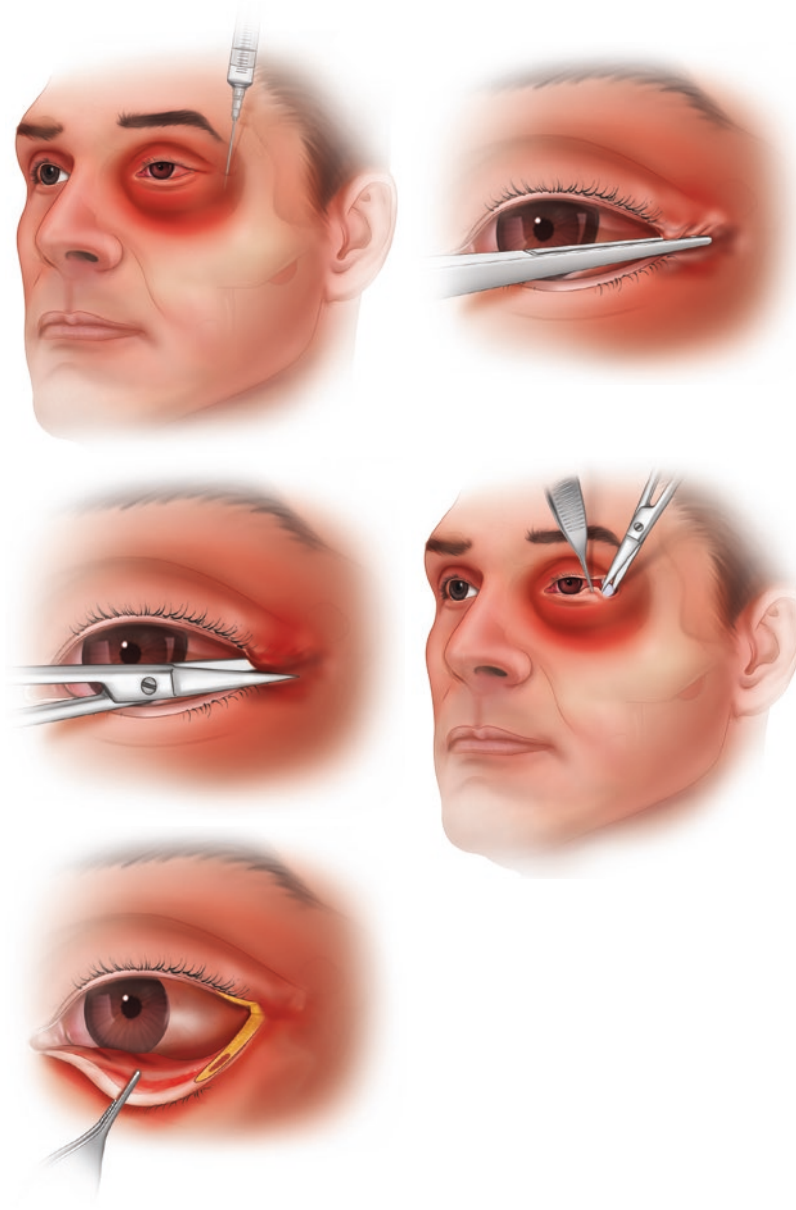

Fig. 13.6 Lateral canthotomy and cantholysis. In the event of orbital compartment syndrome, urgent decompression is needed to prevent loss of vision. The procedure cannot wait for evacuation to ophthalmology. (From [39]. © 2018 Office of the Surgeon General, Borden Institute)

U.S. Forces, Afghanistan, further emphasized the importance of eye protection in all combat troops [25]. Medical personnel and unit leaders should be aware that not all polycarbonate eyewear models meet stringent military antiballistic testing standards and that counterfeit models can be purchased. Military-approved protective eyewear undergoes more extensive testing than commercial equivalents and must be recertified routinely. A list of currently approved eye protection is contained in the Authorized Protective Eyewear List (APEL, available at http://www.peosoldier.army.mil/equipment/eyewear/) [23]. An important aspect of this critical preventive

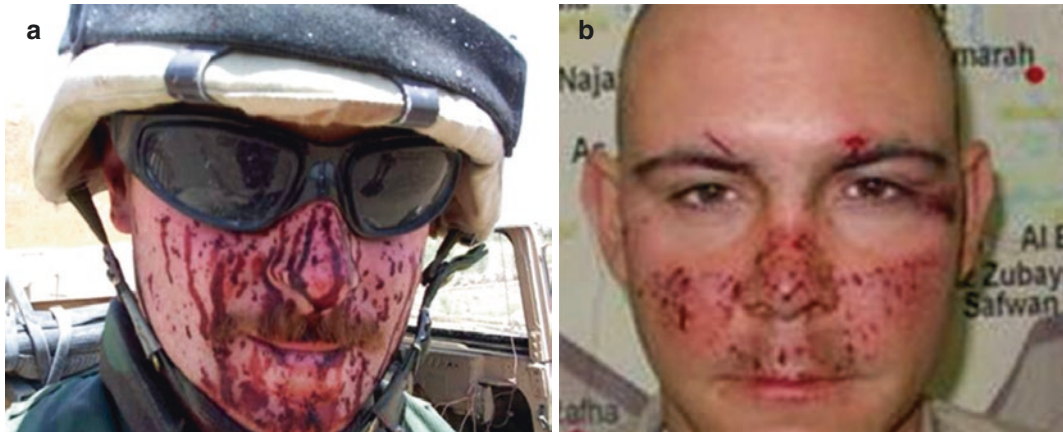

Fig. 13.7 Eye protection. (**a**) Polycarbonate eye protection works but only if it is worn. Eyepro should be considered part of basic field equipment and should be worn both in garrison and in deployed environments. Note the shadow of protection from small fragments demonstrated in (**b**); if any portion of the eye in this shadow zone is injured, presume a significant eye injury exists. Be especially suspicious of penetrating injuries: *shield and ship*. (Courtesy of Dr. Robert A. Mazzoli, MD, FACS)

measure will be to ensure protective eyewear is enforced in garrison and peacetime activities, not just in the combat zone (Fig. 13.7).

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References

1. LaPiana FG, Ward TP. The development of eye armor for the American infantryman. Ophthalmol Clin N Am. 1999;12:421–34.
2. Belkin M, Treister G, Dotan S. Eye injuries and ocular protection in the Lebanon war, 1982. Isr J Med Sci. 1984;20:333–8.
3. Gondusky JS, Reiter MP. Protecting military convoys in Iraq: an examination of battle injuries sustained by a mechanized battalion during operation Iraqi freedom II. Mil Med. 2005;170:546–9.
4. Thomas R, McManus JG, Johnson A, et al. Ocular injury reduction from ocular protection use in current combat operations. J Trauma. 2009;66a:S99–S103.
5. Parker P, Mossadegh S, McCrory C. A comparison of the IED-related eye injury rate in ANSF and ISAF forces at the UK R3 Hospital, Camp Bastion, 2013 (Letter). J R Army Med Corps. 2014;160:73–4.
6. Yonekawa Y, Hacker HD, Lehman RE, et al. Ocular blast injuries in mass casualty incidents; the Marathon bombing in Boston, Massachusetts, and the fertilizer plant explosion in West, Tx. Ophthalmology. 2014;121:1670–6.
7. Lo M, Capo-Aponte JE, Wise D, Simpson D, et al. Ocular battle injuries among U.S. military personnel, 2002–2011. United States Army Aeromedical Research Laboratory report #2013–12; April 2013.
8. Office of The Surgeon General of the Army. Dismounted complex blast injury: report of the army dismounted complex blast injury task force; 2011.
9. Institute of Medicine (IOM). Gulf war and health, volume 9: long-term effects of blast exposures. Washington, DC: National Academies Press; 2014.
10. Cho RI, Bakken HE, Reynolds ME, et al. Concomitant cranial and ocular combat injuries during operation Iraqi freedom. J Trauma. 2009;6:516–20.
11. Weichel ED, Colyer MH, Ludlow SE, Bower KS, Eiseman AS. Combat ocular trauma visual outcomes during operations Iraqi and enduring freedom. Ophthalmology. 2008;115:2235–45.
12. Cockerham GC. Blunt trauma and nonpenetrating injuries of the anterior segment. In: Thach AB, editor. Ophthalmic care of the combat casualty; the textbook of military medicine. Washington DC: Office of The Surgeon General of the United States Army, US Government Printing Office; 2003. p. 137–48.
13. Cockerham GC, Rice TA, Hewes EH, et al. Closed-eye ocular injuries in the Iraq and Afghanistan wars. N Engl J Med. 2011;364:2172–3.
14. Cockerham GC, Lemke S, Rice TA, et al. Closed-globe injuries of the ocular surface associated with combat blast exposure. Ophthalmology. 2014;121:2165–72.

15. Mader TH, Aragones JV, Chandler AC, et al. Ocular and ocular adnexal injuries treated by United States military ophthalmologists during Operations Desert Shield and Desert Storm. Ophthalmology. 1993;100:1462–7.
16. DeSchweinitz GE. Concerning concussion and contusion injuries of the eye in warfare. Am J Ophthalmol. 1919;2:313–9.
17. Tooke F. An experience through the Halifax disaster. Am J Ophthalmol. 1918;1:223–31.
18. Campbell DR. Ophthalmic casualties resulting from air raids. Br Med J. 1941;1:966.
19. Doherty WB. Some of the most important ocular and orbital wounds in war. Am J Ophthalmol. 1942;25:135–48.
20. McAlister CN, Murray TJ, Lakoska H, Maxner CE. The Halifax disaster (1917): eye injuries and their care. Br J Ophthalmol. 2007;91:832–5.
21. Sherwood D, Sponsel WE, Lund BJ, et al. Anatomical manifestations of primary blast ocular trauma observed in a postmortem porcine model. Invest Ophthalmol Vis Sci. 2014;55:1124–32.
22. Butler FK, Giebner SD, Mcswain N, Pons P, eds: Prehospital trauma life support manual.; 8th edn – Military Version 2014.
23. Joint trauma system clinical practice guidelines: initial care of ocular and adnexal injuries 24 Nov 2014 ZTS.
24. Butler FK. The eye in the wilderness. In: Auerbach PS, editor. Wilderness medicine. 6th ed. St Louis: Mosby; 2012.
25. U.S. Forces, Afghanistan (USFOR-A) message 13–067, date/time/group 291018Z Dec 13.
26. All Army Activities (ALARACT) message 176–2008: prevention and treatment of eye injuries in the combat zone.
27. Woodson J. Treatment of traumatic eye injuries: assistant secretary of Defense for Health Affairs memo 14–017 of 7 July 2014.
28. Mazzoli RA, Gross KR, Butler FK. The use of rigid eye shields (Fox shields) at the point of injury for ocular trauma in Afghanistan. J Trauma Acute Care Surg. 2014;77:S156–62.
29. Sauer SA. Management of eye injuries in Afghanistan 2013. CoTCCC presentation; Feb 2014.
30. Brunstetter T, Wasner C, Hart S, Burrow S. Rigid eye shields: a critical gap in the individual first aid kit. J Spec Oper Med. 2013;13:26–8.
31. Calvano C. Rigid eye shields: a critical gap in the individual first aid kit commentary. J Spec Oper Med. 2013;13:29–30 Commentary.
32. Woodson J. Department of Defense Joint-Service first aid kit standardization guidance: assistant secretary of Defense for Health Affairs memo 14-016 of 18 August 2014.
33. Sweet PH 3rd. Occult intraocular trauma: evaluation of the eye in an austere environment. J Emerg Med. 2013;44:e295–e 298.
34. Ritchie JV, Horne ST, Perry J, Gay D. Ultrasound triage of ocular blast injury in the military emergency department. Mil Med. 2012;177:174–8.
35. Debiec M, Frazier T, Colyer M, et al. Inappropriate use of ultrasound in ocular trauma (Letter). Mil Med. 2012;177:v–vi; author reply vi.
36. Blice JP. Imaging of ocular and adnexal trauma. In: Thach AB, editor. Ophthalmic care of the combat casualty; the textbook of military medicine. Washington DC: Office of The Surgeon General of the United States Army, US Government Printing Office; 2003. p. 61–76.
37. Mazzoli RA, Ainbinder DJ, Hansen EA. Orbital trauma. In: Thach AB, editor. Ophthalmic care of the combat casualty; the textbook of military medicine. Washington DC: Office of The Surgeon General of the United States Army, US Government Printing Office; 2003. p. 335–83.
38. Corneal perforation. In Nessen SC, Lounsbury DE, Hetz SP, editors. War surgery in Afghanistan and Iraq; a series of cases, 2003–2007. Washington DC: Office of The Surgeon General of the United States Army, US Government Printing Office; 2008. p. 61–64.
39. Emergency war surgery: 5th United States Revision, 2018. Washington DC: Office of The Surgeon General of the United States Army, US Government Printing Office; 2018.
40. O'Connor K, Butler FK. Antibiotics in tactical combat casualty care 2002. Mil Med. 2003;168:911.
41. Hospenthal DR, Murray CK, Andersen RC, et al. Guidelines for the prevention of infection after combat-related injuries. J Trauma. 2008;64: S211–20.
42. Hospenthal DR, Murray CK, Andersen RC, et al. Guidelines for the prevention of infections associated with combat-related injuries: 2011 update. J Trauma. 2011;71:S210–34.
43. Bhagat N, Nagori S, Zarbin M. Post-traumatic infectious endophthalmitis. Surv Ophthalmol. 2011;56:214–51.
44. Essex RW, Qing Y, Charles PGP, Allen PJ. Post-traumatic endophthalmitis. Ophthalmology. 2004;111:2015–22.
45. Long C, Liu B, Xu C, Yuan Z, Lin X. Causative organisms of post-traumatic endophthalmitis: a 20-year retrospective study. BMC Ophthalmol. 2014;14:34.
46. Colyer MH, Weber ED, Weichel ED, et al. Delayed intraocular foreign body removal without endophthalmitis during operations Iraqi freedom and enduring freedom. Ophthalmology. 2007;114:1439–7.
47. Mader TH, Carroll RD, Slade CS, et al. Ocular war injuries of the Iraqi insurgency, January- September 2004. Ophthalmology. 2006;113:97–104.
48. Harding TH, Statz JK, Williams ST, Martin JS. Blast wave pressure dynamics at the cornea as a function of eye protection. Poster presentation #1034 at the Military health system research symposium 2014, Ft Lauderdale, FL.
49. Breeze J, Allanson-Bailey LS, Hunt NC, et al. Surface wound mapping of occulo-facial injury. Injury. 2012;43:1856–60.
50. Scott R. The injured eye. Phil Trans R Soc B. 2011;366:251–60.

51. Scott GI, Michaelson IC. An analysis and follow-up of 301 cases of battle casualty injury to the eyes. Br J Ophthalmol. 1946;30:42–55.
52. Lew HL, Garvert DW, Pogoda TK, Pei-Te H, et al. Auditory and visual impairments in patients with blast-related traumatic brain injury: effect of dual sensory impairment on Functional Independence Measure. J Rehabil Res Dev. 2009;46:819–26.
53. Goodrich GL, Kirby J, Cockerham G, et al. Visual function in patients of a polytrauma rehabilitation center: a descriptive study. J Rehabil Res Dev. 2007;44:929–36.
54. Cockerham GC, Goodrich GL, Weichel ED, et al. Eye and visual function in traumatic brain injury. J Rehabil Res Dev. 2009;46:811–8.
55. Smith HE. Accommodative defect following atmospheric concussion. Am J Ophthalmol. 1949;32:959–66.
56. Wade AL, Dye JL, Mohrle CR, Galarneau MR. Head, face, and neck injuries during operation Iraqi freedom II: results from the US Navy-Marine Corps combat trauma registry. J Trauma. 2007;63:836–40.
57. Mazur DO. Blunt injury of the posterior segment. In: Thach AB, editor. Ophthalmic care of the combat casualty; the textbook of military medicine. Washington DC: Office of The Surgeon General of the United States Army, US Government Printing Office; 2003. p. 195–210.
58. Hudson SJ. Eye injuries from laser exposure: a review. Aviat Space Environ Med. 1998;69:519–24.
59. FAA Reported Laser Incidents for 2010–2014 (1/13/2015). Available at https://www.faa.gov/about/initiatives/lasers/laws/. Accessed 1 Mar 2015.
60. FAA laser safety initiative. 25 June 2014. Available at http://www.faa.gov/about/initiatives/lasers/. Accessed 1 Mar 2015.
61. FAA fact sheet: laser strikes. 2014. Available at https://www.faa.gov/news/fact_sheets/news_story.cfm?newsId=15774. Accessed 1 Mar 2015.
62. FAA legal interpretation of 14 CFR 91.11, Interference with a Crewmember via laser. 2011. Available at https://www.faa.gov/news/media/Laser%20Memorandum%20Final%20060111.pdf. Accessed 1 Mar 2015.
63. Hollifield RD. Ocular laser injuries. In: Thach AB, editor. Ophthalmic care of the combat casualty; The textbook of military medicine. Washington DC: Office of The Surgeon General of the United States Army, US Government Printing Office; 2003. p. 431–9.
64. All Army Activities (ALARACT) message 144–2008: increase of laser eye incidents.
65. Barkana Y, Belkin M. Laser eye injuries. Surv Ophthalmol. 2000;44:459–78.
66. Brown J, Hacker H, Schuschereba ST, Zwick H, et al. Steroidal and nonsteroidal anti-inflammatory medications can improve photoreceptor survival after laser retinal photocoagulation. Ophthalmology. 2007;114:1876–83.
67. Wyrsch S, Baenninger P, Schmid MK. Retinal injuries from a handheld laser pointer. N Engl J Med. 2010;363:1089–91. Available at http://www.nejm.org/doi/pdf/10.1056/NEJMc1005818. Accessed 1 Mar 2015
68. McLin LN. A case study of a bilateral femtosecond laser injury. 2013. Paper #904 Available at. http://www-conf.slac.stanford.edu/lsow/2013-presentations/LMcLin-RecentFemtosecondLaserInjury-Paper.pdf. Accessed 1 Mar 2015.
69. DoD laser and EMF injury hotline.US Army Combat Readiness/Safety Center CP-12 Safety and Occupational Health newsletter, Winter 2013. Available at https://safety.army.mil/Portals/0/Documents/CP-12/CP-12NEWSLETTER/Standard/CP12_Newsletter_Winter_13Dec2013.pdf. Accessed 1 Mar 2015.
70. Cubano MA, Lenhart MK, Bailey JA, Costanzo GP, Eastridge BJ, Ficke JR, Hults CM, Stockinger ZT, editors. Emergency war surgery. Fourth United States Revision. Washington, DC: Borden Institute; 2013. p. 205–21.
71. Seiff SR. Atmospheric pressure changes and the orbit: recommendations for patients after orbital trauma or surgery. Ophthalmic Plast Reconstr Surg. 2002;18:239–41.
72. Hurst J, Johnson D, Campbell R, Baxter S, Kratky V. Orbital compartment syndrome in a burn patient without aggressive fluid resuscitation. Orbit. 2014;33:375–7.
73. Sullivan SR, Ahmadi AJ, Singh CN, Sires BS, Engrav LH, Gibran NS, et al. Elevated orbital pressure: another untoward effect of massive resuscitation after burn injury. J Trauma. 2006;60:72–6.
74. Ballard SR, Enzenauer RW, O'Donnell T, et al. Emergency lateral canthotomy and cantholysis: a simple procedure to preserve vision from sight-threatening orbital hemorrhage. J Spec Oper Med. 2009;9:26.
75. Breeze J, Horsfall I, Hepper A, Clasper J. Face, neck, and eye protection: adapting body Armour to counter the changing patterns of injuries on the battlefield. Br J Oral Maxillofac Surg. 2011;49:602–6.

14 Ocular Toxicology in Military and Civilian Disaster Environments

Derek L. Eisnor and Brent W. Morgan

Introduction

Visual proficiency is essential to the efficient function of any soldier in theater. Chemical injuries to the eye are true emergencies that necessitate prompt recognition and effective treatment.

The use of chemical weapons (CW) in warfare is well documented throughout history, but not until the industrial revolution did the mass production and deployment of chemical agents become a reality. Most would consider the Germans' effective use of chlorine gas at Ypres, Belgium, in 1915 as the birth of modern chemical warfare. The use of these chemicals including phosgene, sulfur mustard, and lewisites caused nearly 100,000 deaths and over 1 million casualties during World War I (WWI) [1]. Building on the Versailles Treaty of 1919, the Geneva Gas Protocol of 1925 restated the prohibitions previously laid on the wartime use of chemical weapons and added a ban on bacteriological warfare. The United States finally ratified the Geneva Gas Protocol in 1975. In April of the same year, President Ford signed an executive order to prohibit the use of riot control agents (RCAs) in war, except in defensive modes to save lives. Today, dispersal is only allowed in US military operations by presidential order.

In April 1997, the world's first multilateral chemical disarmament Treaty (Chemical Weapons Convention) went into force [2]. Headquartered in Hague, the Organization for the Prohibition of Chemical Weapons (OPCW) is an independent, autonomous international organization with over 500 staff members. As of today OPCW has 190 Member States working toward a collective goal of preventing the use of chemical weapons, thereby strengthening international security. To this end, the Convention contains four key provisions:

1. Destroying all existing chemical weapons under international verification by the OPCW
2. Monitoring chemical industry to prevent new weapons from re-emerging
3. Providing assistance and protection to States Parties against chemical threats
4. Fostering international cooperation to strengthen implementation of the Convention and promote the peaceful use of chemistry [2]

The treaty requires member signatories to destroy existing chemical agents and chemical weapon production facilities under its jurisdiction. Key components of the treaty include strict inspection and verification of compliance, as well as provisions for protection and force assistance from State Parties if needed.

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Despite these efforts, humanity will continue to suffer the malicious use of chemical agents. The ubiquitous presence of toxic chemicals in today's society, and the ability to effectively weaponize many of these agents, makes them particularly attractive to terrorists. Yet the development of RCAs with improved safety profiles allows for effective and humane riot control measures by police, with little or no resultant morbidity to civilians. The answer lies in ongoing research, not only to better our understanding of these agents but also to address possible future threats. It is essential that medical personnel are familiar with the systemic and ocular effects of chemical exposures, as well as their specific treatments.

"Amat Victoria Curam."

Ocular Anatomy and Pharmacology

The eye is vulnerable to chemical exposure from both external contact and systemic (vascular) absorption. The vast majority of CW exposures occur through external contact and the potential for penetration into the eye. For this reason, all parts of the eye can be affected by chemicals, the limitations mainly subject to the pharmacokinetics of a particular agent.

After contact with the outer surface of the eye, many factors determine the extent of damage and penetration into deeper structures. The first site of contact is the tear film, a three-layered structure of alternating hydrophobic and hydrophilic areas. The outermost area is a thin (0.2 μm) layer of lipids secreted by the meibomian glands. This covers the intermediate aqueous layer (0.5 μm), which is maintained by the lacrimal glands. The deepest layer is a thin mucoid layer (0.1 μm) secreted by goblet cells of the conjunctiva and functions as an interface between the hydrophilic tears and the hydrophobic layer of corneal epithelial cells (Fig. 14.1).

At the corneal epithelium, desmosomes form tight junctions underlying a well-organized stratified squamous multicellular layer. This represents the primary barrier to xenobiotic penetration of the anterior chamber. Only relatively hydrophobic chemicals (lipid soluble) will readily pass through this layer. Below this is Bowman's membrane, which separates the epithelium from the stroma. Much like the alternating layers of the tear film, the stroma is a hydrophilic layer comprising primarily of

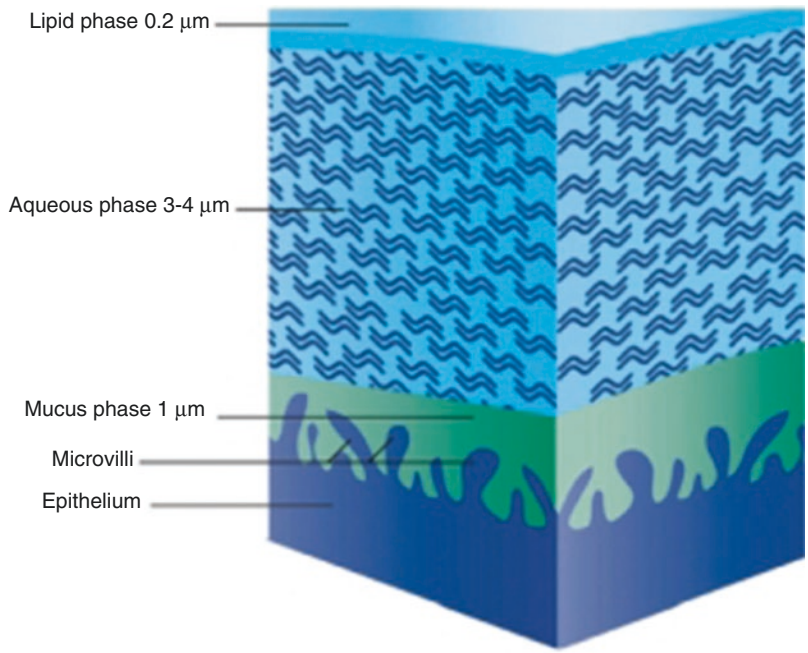


Fig. 14.1 Three components of tear film. Dr. Philip Morgan, Tear film proteins: examining production, role and interaction with contact lenses, http://www.clspectrum.com/articleviewer.aspx?articleID=104177

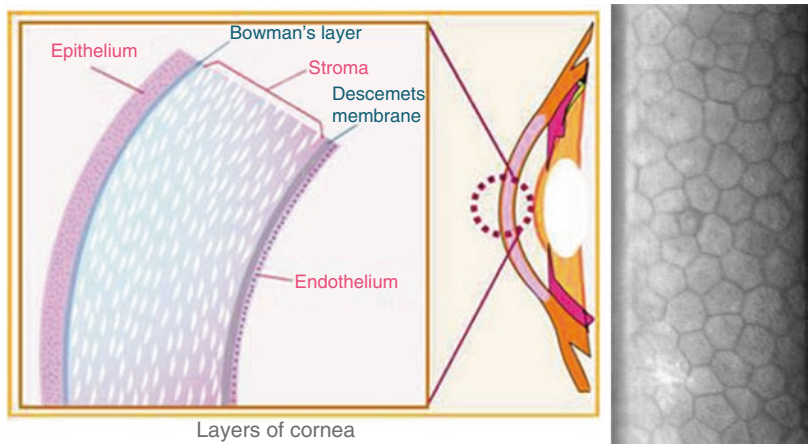


Fig. 14.2 Left: Diagram of the cornea (Janice M. Epstein, New Research Using Regenerated Corneal Cells Shows Positive Results, http://blog.bostonsight.org/wp-content/uploads/2013/12/corneal_endothelium.jpg). Right: corneal endothelium under a microscope. Wikipedia. Professor Yann Gavet. Cornea endothelium specular https://fr.wikipedia.org/wiki/Fichier:Cornea_endothelium_specular.jpg

collagen and glycosaminoglycans. Although it may slow the absorption of lipid-soluble agents, it may also act as a reservoir for water-soluble chemicals, which may prolong damage to these structures (Fig. 14.2).

At the most proximal layer of the stroma is Descemet's membrane, which functions as the basement membrane, secreted by the innermost layer or corneal endothelium. These specialized, mitochondria-rich cells govern fluid and solute transport across the posterior surface of the cornea.

The primary function of the corneal endothelium is to allow for "passive leak" of nutrients, solutes, and glucose from the aqueous to the more superficial structures of the cornea and stroma. At the same time, the endothelium actively pumps water back out from the stroma into the aqueous. This is required to maintain the cornea in the slightly dehydrated state that is required for optical transparency. These processes are heavily dependent on functioning Na/K/ATPase transport mechanisms and the enzymatic activity of carbonic anhydrase.

Damage to limbus structures can lead to limbal stem cell deficiency (LSCD). These progenitor cells function to replace corneal epithelial cells and form a barrier to encroachment by conjunctival epithelium (Fig. 14.3). Loss of function

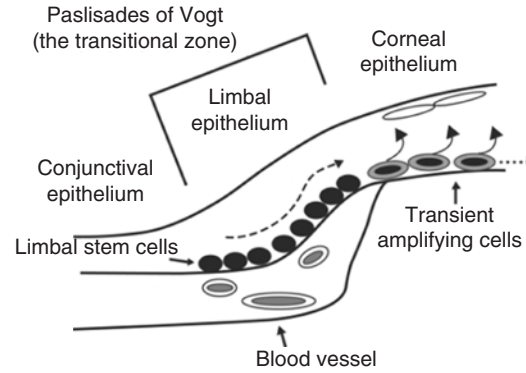


Fig. 14.3 Schematic representation of corneal epithelial cells. (Kayama et al. [203], Copyright © 2007 Dove Medical Press Limited. Open access)

leads to chronic inflammation, scarring, and corneal opacification [3].

Since corneal endothelial cells are postmitotic and rarely divide, damage to the corneal endothelium results in healing via lateral spread and enlargement of adjacent endothelial cells. In severe cases, there is a loss of cellular architecture, leading to irregular cell size and shape variations (polymegathism). This is also accompanied by corneal edema, which disrupts regular spacing between stromal collagen fibrils and causes surface irregularities in the epithelium. The resulting light scatter causes decreased

visual function. Further damage leads to neovascularization, chronic keratitis, and recurrent corneal scarring, causing long-term visual loss. Although typical of blister agents, these injury patterns have been described with higher concentrations of RCAs [4].

Blood supply to the eye comes from the ophthalmic artery (OA), which arises as the first branch off the internal carotid artery (ICA). Branches of the OA can be subdivided into two groups: an orbital group, which includes the lacrimal artery, supplying the orbit, adnexa and surrounding structures, and an ocular group, supplying the muscles and bulb of the eye. The retina has dual arterial supply from both choroidal and retinal vessels. There are tight junctions within the retinal endothelium that function much like those that form the blood-brain barrier in cerebral capillaries. However, at the optic disk, these junctions are lacking, and therefore, hydrophilic molecules can enter the optic nerve by diffusion from the extravascular space [5]. This also represents an area where substances may penetrate into the CNS. The outer retina consists of the retinal pigment epithelium (RPE), rod and cone outer and inner segments, and the outer photoreceptor nuclear layer. This portion of the retina is supplied by the choriocapillaries of the posterior ciliary arteries. These vessels do not possess tight junctions and are highly permeable to large proteins such as albumin [6].

Systemic exposure to chemicals results in distribution to all parts of the eye via uveal and retinal blood vessels. As mentioned above, due to lack of tight junctions, the outer retina and RPE are highly susceptible to hydrophilic molecules less than 200 daltons, which can cross the ciliary and iris capillaries and diffuse into the aqueous humor [6]. This represents the most likely target site for xenobiotic-induced retinal injury.

Melanin is synthesized from tyrosine in melanocytes. In addition to providing pigmentation (eumelanin) for skin, hair, and eyes, melanin is capable of binding chemicals and acts as a buffer against oxidative injury. Melanin has a high binding affinity for heavy metals, aromatic hydrocarbons, and free radicals. Binding to and buffering of harmful agents may limit potential damage at cellular receptor sites. However, the same mechanism may lead to the accumulation and slow release of chemicals, resulting in chronic toxicity [7]. The retinotoxic drug chloroquine accumulates in very high concentrations in the choroid, RPE, iris, and ciliary body due to its affinity for melanin [8].

The retina is a multilayered structure that occupies the inner lining of the posterior globe. The retina and optic nerve developmentally are considered part of the CNS [9]. Visual images transmitted from the cornea and lens are projected onto the retina, initiating a series of neurochemical events leading to the transmission of neural impulses from retinal ganglion cells across the optic nerve to the brain.

Interpretation of light stimulus is mediated by two main groups of specialized photoreceptive neurons: rods and cones. Below a certain luminescent level, night vision (scotopic) is mediated by rods. At higher luminescent levels, cones provide predominant function over daylight (photopic) vision. The range where two mechanisms are working together is called the mesopic range.

The retina comprises many layers of specialized cell types. From anterior to posterior orientation, first is the basement membrane formed by Muller cells, which help separate the nerve fiber layer from the ganglion (nerve cell bodies) layer. Beyond this are the inner nuclear and outer plexiform layers, which synapse with the dendrites of bipolar cells (which transmit signals from photoreceptors and pass on to ganglion cells). Finally, the outer nuclear layer, which contains the photoreceptor cell bodies (rods and cones) and the external limiting membrane (separates the photoreceptor layer from their cell bodies). Last or innermost is the rod/cone photoreceptor layer and the retinal pigmented epithelium (RPE). It is counterintuitive how light must pass through several outer layers to reach the photoreceptor layer.

The optic disc (at the optic papilla) is where optic nerve fibers leave the eye and is devoid of photoreceptors (AKA the blind spot). Temporally opposite to this is the macula, which contains the highest density of photoreceptors. At its center is the fovea, which is responsible for sharp central vision.

Metabolism of xenobiotics occurs throughout the eye via phase I and phase II biotransformation reactions. Specific drug-metabolizing enzymes such as acetylcholinesterase, carboxylesterase, alcohol and aldehyde dehydrogenases, aldehyde and aldose reductases, superoxide dismutase, and monoamine oxidase are some of the proteases represented in the ciliary body and tears, choroid, and retina. A number of cytochrome P450 enzymes are present in various ocular tissues, and most are represented in the retina [10]. Apart from the lens, ocular tissues such as the cornea, choroid, and retina contain the most biotransformation enzymes including phase II enzymes. This is intuitive given that these tissues have high rates of metabolism and regularly interact with both lipid- and water-soluble xenobiotics and free radical forming UV radiation.

Injury Patterns

Over the last 20 years, we have extensive data of ocular injury patterns from our military efforts in the Middle East. This has led to significant advances in prehospital management of trauma patients. Apart from conventional weapons, the regular use of improvised explosive devices (IEDs) has caused devastating ocular injuries. In an observational study of ocular war injuries during the Iraqi insurgency, blast fragmentations from munitions accounted for over 80% of all injuries, and the most common single cause of injury (51%) was IEDs [11].

Explosives may be categorized as high-order (HE) or low-order explosives (LE). HE produce a defining supersonic overpressurization shock wave, and examples include TNT, C-4, Semtex, nitroglycerin, dynamite, and ammonium nitrate fuel oil (ANFO). LE create a subsonic explosion, which lacks the overpressurization wave. Examples include pipe bombs, gunpowder, and most pure petroleum-based bombs [12]. Common devices used in recent civilian incidents consist of a HE core packed in oil products and a variety of shrapnel.

Explosions cause a variety of injuries by different mechanisms. A positive phase is marked by a massive increase in atmospheric pressure or blast wave that frequently produces injuries to the lungs, CNS, gastrointestinal, auditory, and ocular systems. Open globe injuries in this setting are complicated and result in complete surgical excision in more than half of cases. A negative phase follows, in which there is a drop in atmospheric pressure, which draws debris into the blast area. Secondary penetrating injuries from explosive debris as well as thermal injuries are common. Unlike isolated projectile injuries, these injury patterns present a unique challenge for rapid and accurate triage of victims. In addition to type, composition, and amount of explosives used, victim locations relative to the blast, enclosed space, and the presence of protective equipment or barriers can help determine the severity of injuries sustained. Ongoing research in the use of colorimetric blast dosimeters, which change color in the presence of a blast exposure, may help more rapidly identify victims of threshold ocular and brain injuries [13].

Gross contaminants from secondary projectiles can commonly complicate traumatic injuries. In addition to a variety of debris causing penetrating injuries, tissue damage may be compounded with exposure to chemical agents, causing extensive burns or direct chemical injury. Some IEDs have been compounded with jellied gasoline or diesel fuel mixed with ammonium nitrate (AN-FO) causing severe burns. Often planted in trees, resulting blast injuries may also be contaminated with biological debris. Some of these injuries may have been prevented by the use of protective eyewear. Only a minority of cases reported using protective lenses at the time of injury [11]. Enhanced vehicle shielding particular to IEDs, as well as improvements in protective lenses, will no doubt help to reduce the morbidity associated with these enemy devices.

Apart from open globe trauma, periorbital hemorrhages second-degree burns to adnexa and corneal injuries are common. Traumatic optic neuropathy (TON), although not as frequent, is an ominous injury and should be suspected in any patient presenting with decreased visual acuity or a relative afferent pupillary defect (RAPD). Thermal burn injuries involving the cornea frequently have moderate visual loss at follow-

up. In studies looking at firework injuries, corneal injuries accounted for almost 40% (16 of 42 patients), and as many as 31% had moderate-to-severe visual loss at 3-month follow-up, with evidence of corneal scarring [13]. A similar series of firework injury pattern reported corneal injuries as the most common, and as many as 28% (32 of 116 patients) had permanent visual loss secondary to corneal or retinal injury [14].

Today, isolated battlefield chemical ocular injuries are rare, and thus, the majority of our understanding in the areas of chemical-induced injury patterns is derived from animal models. Injuries are largely dependent on exposure concentration or dose, time of exposure, and composition of the specific chemical agent. Historically, the effects of mustard agents are well known. Ocular injuries appear in over 75% of all mustard gas casualties with reports of delayed ocular morbidity including keratopathy, stromal keratitis, recurrent corneal erosions, and permanent blindness years later [15].

Chemical Injuries

Chemical exposures to the eyes represent a true ophthalmic emergency. Injuries occur both on the battlefield and during peacetime operations, and treatment principles are very similar. As soon as ocular exposures are suspected, immediate and copious irrigation to the eyes is indicated with any neutral solution available. Regardless of the particular xenobiotic, the foremost priority is rapid dilution of the chemical exposure. The timing and efficiency of this process cannot be overstated and may be vision-saving.

Chemical warfare (CW) agents have varying degrees of local and systemic toxicity. Some exposures may present with delayed toxicity. Familiarity of these effects, early signs and symptoms, and treatment priorities are essential to all battlefield medics. Operators should maintain a high index of suspicion concerning the use of chemical warfare agents in the presence of multiple casualties within a finite time and locale. Our discussion will focus specifically on ophthalmic injuries with these and other agents. Structured approaches for general medical interventions to exposures from CW agents, as well potential natural and terrorist disasters, are outlined in comprehensive preparedness courses such as TERT (Technical Emergency Response Training) for CBRNE (Chemical, Biological, Radiological, Nuclear and Explosive) incidents or AHLS (Advanced Hazmat Life Support).

The potential for ocular chemical exposures is everywhere. Common occupational hazards that can cause significant eye injuries include acids (e.g., refrigerants, car battery acids, and pool cleaning chemicals) and alkalis (e.g., drain cleaners, household cleaning supplies, ammonia, fertilizers, and building supplies). Eye protection is most effective in injury prevention when practiced.

In the training environment, both military and law enforcement are exposed to various riot control agents (RCA). These agents are classified as tear agents or lachrymators and quickly cause irritant effects to eyes, skin, and mucosal membranes. When used within confined spaces, these agents have been shown to induce significant edema and chemosis with loss of corneal epithelium [16]. Permanent injuries have been attributed to close dispersal via direct spray or grenade mechanisms [17]. There is also risk of thermal and explosion injuries in these training scenarios.

Acids are defined as chemical substances in aqueous solution that react with bases and certain metals to produce salts. In solution, they have a pH of less than 7, which reflects a higher concentration of hydrogen ions (protons). Normal physiologic pH of the human eye is nearly 7.4. Significant damage to ocular structures usually occurs from solutions with a pH of less than 4. Contact with ocular surfaces results in corrosive destruction via coagulative necrosis. Cellular injury results in precipitation of tissue proteins, creating a barrier to further penetration. Sulfuric acid from car batteries frequently causes significant damage to the conjunctiva and corneal epithelium.

Compared to alkaline solutions, acids do not typically cause deep penetrating injuries. Hydrofluoric acid (used in rust removers and glass etching) is one of a few notable exceptions.

Hydrofluoric acid (HFA) rapidly penetrates and destroys the corneal endothelium. Latent complications include epithelial erosions, keratoconjunctivitis, symblepharon, and progressive vascularization of the cornea, leading to scarring and decreased visual function. Often, anterior chamber paracentesis and irrigation are required to limit further injury [18]. Damage is largely due to fluoride ion toxicity, which binds calcium and can lead to life-threatening hypocalcemia. Depending on the concentration of HFA, this can result with total body surface area (TBSA) exposures of less than 3%. Following rapid and copious irrigation, many authors advocate eye instillation with calcium gluconate drops, although clinical research in this area is lacking. Recommended concentrations vary between 1% and 10%, and some advocate drops every 2–3 hours for several days [19].

Alkalis are ionic salts that dissolve in water to form base solutions (pH greater than 7). In general, these agents cause more damage than acids due to extensive penetration of body tissues. The corrosive damage by liquefactive necrosis results in consumption of cell membranes and release of proteolytic enzymes, enhancing further injury. Permanent visual loss frequently results from agents with pH > 11, and tissue damage continues until the offending agents are consumed. Penetration into the anterior chamber can be rapid (<15 seconds), and damage to limbal stem cells frequently results in permanent corneal injury and opacification. These progenitor cells function to replace corneal epithelial cells and form a barrier to encroachment by conjunctival epithelium. In addition, secondary glaucoma can result from damage to the trabecular meshwork and scarring contraction of the anterior globe [20]. Advances in treatment options, including amniotic membrane transplantation (AMT), autologous limbal stem cell transplantation (ALT), with or without penetrating keratoprosthesis (PKP), continue to show promise in restoring vision, but results are highly variable [21, 22].

Anterior structures of the eye often suffer catastrophic damage to alkali burns. Despite advances in surgical techniques to restore anterior media transparency, damage to posterior structures is not easily appreciated. Postoperative glaucomatous changes can result in optic nerve damage. Monitoring has proved particularly challenging, with no reliable way of checking intraocular pressure (IOP) to date. Some surgeons will place a tube shunt at the time of keratoprosthesis placement in anticipation of this problem [22]. Even in the setting of normal IOP, premature vision loss is noted. Cade et al. studied the effects of anti-tumor necrosis factor alpha (TNF-a) antibodies on inflammatory cytokines with alkali-induced corneal burns in mice. Besides expected corneal injury, histopathology revealed retinal damage with apoptosis to the RGC within 24 hours and a second wave at 1-week post injury. A single dose of anti-TNF-a antibodies demonstrated a marked reduction in inflammatory cytokines, as well as a reduction in corneal neovascularization [23]. Further investigation in these areas is needed to better delineate these complex injury mechanisms.

It cannot be overstated that once a chemical exposure to the eye is suspected, nothing should delay immediate and copious irrigation with any neutral solution available (e.g., water, milk, iced tea, and saline solution). Following pH neutralization and screening for possible open globe injuries, further evaluation may proceed for extent of injury and quantification of visual impairment.

There are several classification schemes for ocular chemical injuries, including Hughes, Thoft, Roper-Hall (Table 14.1), and Dua grad-

Table 14.1 Roper-Hall classification of ocular chemical injuries

	Clinical findings		
Grade	Cornea	Conjunctiva limbus	Prognosis
I	Corneal epithelial damage	No limbal ischemia	Good
II	Corneal haze, iris details visible	<1/3 limbal ischemia	Good
III	Total epithelial loss, stromal haze, and iris details obscured	1/3–1/2 limbal ischemia	Guarded
IV	Cornea opaque, iris, and pupil obscured	>1/2 limbal ischemia	Poor

Adapted from Roper-Hall [201]

ing systems. Regardless of which system is used, careful attention to the extent of corneal, limbal, and conjunctival damage is paramount to accurate grading and treatment planning. The degree of limbal ischemia (number of clock hours) and depth of corneal injury are vital prognostic factors, as well as any evidence of increased IOP.

Grade 2 injuries display only focal limbal ischemia and mild corneal haze but may develop neovascularization (scarring) at the site of stem cell loss. Grade 3 injuries show ischemia of most of the limbus with significant corneal haze, limiting visualization of anterior chamber structures. Often the corneal epithelium undergoes encroachment by conjunctival epithelium with a significant decrease in visual acuity usually requiring surgical intervention.

Grade 4 injuries represent near or total loss of limbal stem cells and destruction of proximal conjunctival epithelium. The cornea is completely porcelainized. Return of vision may not be possible without aggressive surgical management.

In patients with severe corneal injury (Roper-Hall IV), the Dua system (Table 14.2) provides further subclassification of severe injuries (IV–VI) to more accurately predict outcomes and help guide interventions [24].

Table 14.2 Dua classification of ocular chemical injuries

Grade	Clinical findings	Conjunctival involvement	Prognosis
I	0 clock hours of limbal involvement	0%	Very good
II	<3 clock hours of limbal involvement	<30%	Good
III	Between 3 and 6 clock hours of limbal involvement	30–50%	Good
IV	Between 6 and 9 clock hours of limbal involvement	50–75%	Good to guarded
V	Between 9 and 12 clock hours of limbal involvement	75–100%	Guarded to poor
VI	Total limbus involved	Total conjunctival involvement	Very poor

Adapted from Dua et al. [25]

Early re-epithelialization is the most important determinant of ultimate visual function. Corneal epithelial regeneration serves to protect against proteolytic enzymes contained within the tear film. These enzymes are in contact with exposed stromal tissues that are susceptible to further damage and, if unchecked, may lead to corneal perforation.

The inflammatory response to chemical injury begins immediately and can hinder organized re-epithelialization. Regimented use of corticosteroids and immune modulators is essential in the acute phase of healing to limit these processes. Changes in inflammation, corneal clarity, and IOP should be monitored closely (daily) as indicators of response to interventions and ultimate prognosis.

Long-acting cycloplegics should be used for comfort along with oral pain medications. High-dose vitamin C is protective against both UV-based and chemical-induced inflammatory changes with acute corneal injury [25–27]. Broad-spectrum antibiotics are indicated as well as a tetracycline derivative (doxycycline) for both antimicrobial and anti-inflammatory effects.

Enzenauer et al. demonstrated the synergistic effects of silibinin, doxycycline, and dexamethasone in reducing inflammation-mediated vesicant-induced ocular injuries [28]. These injuries induce inflammatory changes in the epithelium; apoptotic cell death; and increased expression of vascular endothelial growth factor (VEGF), cyclooxygenase-2 (COX-2), and matrix metalloproteinase-9 (MMP-9) in cultured rabbit eye corneas. Doxycycline has the ability to inhibit MMPs, and dexamethasone effectively reduces VEGF levels. The combination of these agents together with silibinin effectively reduced all injury-associated endpoints including cell death, micro bullae formation, neovascularization, MMP-9 and COX-2 levels, and epithelial thinning.

The inflammatory response in the first week after injury (acute phase) determines transition to chronic inflammation, stromal repair, and scar

tissue formation. Ideally, at this point, corneal re-epithelialization is complete, and steroids are tapered. Ongoing inflammation in the setting of persistent corneal defects usually requires cautious extension of steroids, given the increased risk of perforation if used beyond 21 days [29]. Due to these limitations, failure of significant re-epithelialization within 10–14 days (early healing phase) post injury should prompt consideration of surgical intervention.

Beyond 3 weeks (late healing phase), maintenance of normal stromal function, close monitoring of IOP, and continued cornea care with topical lubricants and tear substitutes are vital. Loss of corneal sensation and damage to mucin-producing cells lead to inadequate corneal tear film production. Surgical evaluation is indicated for any persistent epithelial defects, and tarsorrhaphy may be indicated [30].

Ocular surface irritation or injury is typically measured by Draize testing, which has been the standard process for over 50 years. Chemist and Doctor of Pharmacology, John Henry Draize was recruited by the US Army in 1935 to investigate the effects of mustard gas and other chemical agents. Testing involves instillation of a fixed amount of liquid or solid xenobiotic into the conjunctival sac of one eye and closure of the eyelids for 1 second. Both the treated and untreated eyes are closely evaluated over the next several days. Changes to the cornea, conjunctiva, and lens are noted on a standard scale with a maximum score of 110. Scale weighting favors corneal injuries (maximum 80), as they have significant impact on visual function. A similar test is used to quantify xenobiotic-induced skin irritation.

The Draize test has been criticized for inter-observer variability, limited application of animal response to human eyes, and causing unnecessary cruelty to animals. Draize testing, developed for humanitarian purposes and highly regulated by federal agencies, has been modified over the years to reduce the number of animals needed for testing and amount/concentration of xenobiotics used. Alternate tests, including hen's egg chorioallantoic membrane, human epidermal keratinocytes, and in vitro human corneal cell cultures, have been studied. To date, no single alternative test has accuracy equivalent to Draize testing; however, a combination of analyses may prove comparable [31–34]. This is not surprising given our limited understanding of injury mechanisms from xenobiotic-induced ocular injuries.

Organic solvents are ubiquitous in modern society. These carbon-based xenobiotics are extremely useful in their ability to dissolve a plethora of materials and are widely used in paints, varnishes, lacquers, adhesives, glues, and degreasing/cleaning agents. They are also used in the production of dyes, polymers, plastics, textiles, printing inks, agricultural products, and pharmaceutical products. Accidental eye exposures frequently cause damage to lipophilic structures, including the corneal epithelium. Most of these agents do not have extremes from physiologic pH, however, they should be treated rapidly as with any acid or alkali injury. Organic solvents rapidly pass through cellular membranes, disrupting cell membrane functions and vital homeostasis mechanisms [35]. Even solvents common to ophthalmic medications, such as dimethyl sulfoxide (DMSO), used as a vehicle for topical application of pharmaceuticals, can cause corneal cellular injury at low doses [36]. Treatment priorities include copious irrigation until adequate decontamination, followed by evaluation for corneal injury. Typically, these injuries heal well without permanent dysfunction or visual impairment.

Similar to solvents, *surfactants* are common compounds that have the ability to emulsify or reduce surface tension between the interfaces of two surfaces. They contain both hydrophobic groups and hydrophilic groups and thus have the ability to mix both water-soluble and water-insoluble agents. Surfactants are frequently used as detergents, dispersal agents, and foaming agents. Their lipophilic terminus allows for rapid penetration through cellular tissues, and the hydrophilic portion allows for concentration in water-soluble tissues (corneal stroma). The hydrophilic terminus may be strongly ionic (and highly reactive) in solution. Typically, cationic agents are more damaging than anionic solutions. Benzalkonium chloride, a cationic detergent, is one of the most commonly used preservatives in topical preparations. At low concentrations

(<0.01%), its surfactant qualities dissolve the lipid phase of the tear film, increasing drug penetration or delivery. At higher concentrations, it exerts direct cellular toxicity via damaging cytoplasmic membranes and cytoplasmic organelles and impeding metabolic cellular function [37].

Personal Protective Equipment (PPE)

All providers should be familiar with the classification and general use of personal protective equipment (PPE). This equipment is designed to protect providers from illness or injury resulting from contact with chemical, radiological, or biologic hazards. Selection of protective equipment is made specific to a given threat response. In scenarios with exposure to unknown agents, a minimum use of class B protection is recommended. No single PPE ensemble can protect the wearer from all hazards.

Appendix B of the HAZWOPER standard defines the OSHA/EPA Protection Levels A, B, and C as follows:

- Level A—To be selected where the hazards are unknown or unquantifiable or when the greatest level of skin, respiratory, and eye protection is required.
- Level B—The highest level of respiratory protection is necessary, but a lesser level of skin protection is needed.
- Level C—The concentration(s) and type(s) of airborne substances is known, and the criteria for using air-purifying respirators are met.

These are general descriptions of protection level only and do not guarantee immunity from CBRN-specific hazards. The HAZWOPER standard itself states that the generic descriptions of the equipment do not fully address the performance of PPE in relationship to specific needs [38].

Specific NIOSH-approved equipment appropriate to each level is as follows:

Level A protection should be worn when the highest level of respiratory, skin, eye, and mucous membrane protection is needed. A typical Level A ensemble includes the following:

- Positive pressure (pressure demand), self-contained breathing apparatus (SCBA) or positive-pressure supplied air respirator with escape SCBA
- Fully encapsulating chemical protective suit
- Gloves, inner and outer, chemical resistant
- Boots, chemical resistant, steel toe, and shank (depending on suit boot construction, worn over or under suit boot)

Level B protection should be selected when the highest level of respiratory protection is needed, but a lesser level of skin and eye protection is needed. Level B protection is the minimum level recommended on initial site entries until the hazards have been further identified and defined by monitoring, sampling, and other reliable methods of analysis and equipment corresponding with those findings utilized. A typical Level B ensemble includes the following:

- Positive-pressure (pressure-demand), self-contained breathing apparatus or positive-pressure supplied air respirator with escape SCBA
- Chemical-resistant clothing (overalls and long-sleeved jacket, coveralls, hooded two-piece chemical splash suit, disposable chemical-resistant coveralls)
- Gloves, inner and outer, chemical resistant
- Boots, outer, chemical resistant, steel toe, and shank

Level C protection should be selected when the type of airborne substance is known, concentration measured, criteria for using air-purifying respirators met, and skin and eye exposure is unlikely. Periodic monitoring of the air must be performed. A typical Level C ensemble includes the following:

- Full-face or half-mask, air-purifying respirator
- Chemical-resistant clothing (one-piece coverall, hooded two-piece chemical splash suit, chemical-resistant hood and apron, disposable chemical-resistant coveralls)

- Gloves, inner and outer, chemical resistant
- Boots, steel toe and shank, chemical resistant

Level D protection is primarily a work uniform and is used for nuisance contamination only. It requires only coveralls and safety shoes/boots. Other PPE is based on the situation (types of gloves). It should not be worn on any site where respiratory or skin hazards exist (Chemical Hazards Emergency Medical Management, DHHS; http://chemm.nlm.nih.gov).

In addition, the National Fire Protection Association (NFPA) has defined protective equipment standards for response to CBRNE terrorist incidents. There are three categories of protective ensemble depending on the hazard type (vapors, liquids, and particulates) and the airborne contaminant level. Selection is based on thorough risk assessment in accordance to OSHA/HAZWOPER regulations:

- Class 2 ensembles are intended for use at terrorism incidents involving vapor or liquid chemical or particulate hazards where the concentrations are at or above IDLH level requiring the use of CBRN compliant self-contained breathing apparatus (SCBA).
- Class 3 ensembles are intended for use at terrorism incidents involving low levels of vapor or liquid chemical or particulate hazards where the concentrations are below IDLH, permitting the use of a CBRN compliant air-purifying respirators (APR) or power air-purifying respirator (PAPR).
- Class 4 ensembles are intended for use at terrorism incidents involving biological or radiological particulate hazards where the concentrations are below IDLH levels permitting the use of CBRN compliant APR or PAPR. The ensembles are not tested for protection against chemical vapor or liquid permeability, gas-tightness, or liquid integrity [38].

Decontamination

Decontamination is accomplished by either physically removing or chemically neutralizing a toxic substance. In order to minimize ongoing injury from exposure to chemical agents, a systematic stepwise approach to decontamination should be familiar to all healthcare providers. For the purpose of our discussion, healthcare "providers" will include all personnel, from first responders and field medics to nurses and emergency/trauma physicians.

The first priority in all exposure scenarios is to avoid secondary contamination or exposure to providers. This is accomplished by appropriate use of personal protective equipment (PPE) by all providers. Effective decontamination is time dependent; however, until the specific chemical agent(s) is known, the highest level of protection (level A or B PPE) is recommended. Decontamination should occur prior to patient transport to a civilian or combat support hospital (CSH); however, this is not always possible. Regardless of use, it is vital to maintain separation of clean and "dirty" or contaminated areas and resources (i.e., vehicles and medical equipment).

In mass casualty events, staging areas need to be established between the exposure area(s) or hot zone and medical treatment areas. These include key areas such as decontamination and triage to effectively assess and treat large numbers of victims. The exact location and access of these areas involve many factors including, but not limited to, the specific exposure agent(s), number of potential victims, wind direction and weather conditions, presence of local shelter, evacuation routes, geography, and local resources.

The second priority is timely decontamination of victims. This is particularly true with skin exposures to liquid chemicals. Rapid decontamination will help minimize adverse health effects and, in turn, utilize less medical resources. If contaminated, carefully remove clothing with attention to possible injuries. If necessary, clothing may be cut and rolled away from the patient in such a fashion as to avoid further skin contact. Clothing should be double bagged in plastic to avoid further exposure and labeled for potential forensic analysis.

General recommendations for skin decontamination include copious irrigation with soap and water. Avoid contamination of unexposed

areas, with particular attention to the face, eyes, and urogenital areas or open wounds. Be aware of contaminated water runoff in addition to what may collect in the patient's socks and shoes. Moisture may enhance dermal absorption of certain chemical agents such as mustards. In this setting, the skin should be dusted with Fuller's earth or Canadian Reactive Skin Decontaminant Lotion (RSDL) prior to thorough washing with soap and water [39].

With regard to chemical warfare (CW) agents, chemical decontamination can be much more effective than simple irrigation with water. Chemical decontamination either binds or converts toxic CW agents into innocuous products. Dry formulations that contain Fuller's earth include PDK-1,2 and M-291 kits that are available for decontamination of skin as well as equipment. Diluted hypochlorite (0.5%) solution (one-part household bleach with ten parts water) is effective against mustards and V-nerve agents but not G-agents [40, 41]. Ready-to-use chemical formulations such as DS2 are commercially available. The active ingredient is ethylene glycol monomethylether, which hydrolyzes CW agents into nontoxic products. Other kits available include the M258A1, M280 (American), and C8 (German), which contain variable concentrations of sodium hydroxide or calcium hypochlorite. Ideal characteristics of decontamination agents are water-solubility, harmless to humans, stable for long-term storage and rapidly effective against most potential contaminates.

Chemical exposures to the eye require immediate irrigation to help minimize vision-threatening injuries. Whether secondary to occupational accident or on the battlefield, the key is prompt recognition and intervention. Most exposures occur far from a hospital setting, making prehospital treatment crucial in the severity and potential long-term disability from such injuries. Copious irrigation with tap water or isotonic saline has long been the standard of treatment for chemical eye exposures. Animal studies have shown no significant benefit of isotonic fluid irrigation compared to sterile water [42]. More recent studies demonstrate better outcomes with tap water irrigation of alkali burns, which may be largely due to immediate irrigation at the scene of the injury [43]. Since time from injury to decontamination is critical, prehospital irrigation is often dictated by what is readily available. Standard recommendations include a minimum of 15–20 minutes of irrigation for mild exposures, including dilute acids. This includes exploration and examination of all adnexa for gross contaminants. Continue complete ocular decontamination, including thorough cleansing of fornixes until normalization of tear film pH (7.4–7.6) and symptomatic improvement. More severe exposures including alkali burns often require extensive irrigation for several hours.

Recent studies have looked at using various buffering solutions for irrigation to improve injury outcomes. Diphoterine rinsing solution (DRS) is a polyvalent, hypertonic, neutral pH, amphoteric, water-soluble compound, which has the capacity to bind both acids and bases, oxidizing agents, and solvents [44, 45]. Animal studies looking at corneal injury, IOP elevation, and systemic oxidative stress from exposure to nitrogen mustard found DRS superior to isotonic saline irrigation [46]. In addition to mechanical decontamination, amphoteric compounds function as chelators, binding a variety of xenobiotics and radio nucleotides. Also, its hypertonic properties impede chemical tissue penetration and remove a portion of toxicants within the cornea [44]. Merle et al. (2005) looking at the efficacy of DRS for human ocular alkali burns showed faster corneal healing times with DRS. Skin decontamination studies to mustard agents have shown DRS was more effective than soap and water or isotonic saline [47]. While DRS is currently awaiting FDA approval, alternative agents include the borate-buffered balanced saline solution (Cederroth Eye Wash). Animal studies with NaOH-induced corneal burns showed significant improvement in anterior chamber pH with Cederroth versus saline solution regardless of irrigation rates [48].

Further development in these areas may provide reconstitution of dry products by medics in the field, supplying superior decontamination solutions without compromising team mobility. If buffered solutions are not available, many authors would recommend irrigation with tap water.

Patient Evaluation

Following rapid survey and stabilization of airway, breathing, and circulation, the patient should undergo *immediate* irrigation of the eyes prior to any detailed ocular evaluation in the setting of *suspected* chemical exposure. As mentioned before, the priority should be flushing the eye with what is readily available rather than delay decontamination. Alkali injuries can penetrate the cornea and anterior chamber in seconds to minutes. Ideal eye rinses include buffered solutions particular to the chemical exposure (if known) but seldom are these readily available to the first responder. Tap water is usually available, but any neutral solution is acceptable, including milk or iced tea.

Key historical elements include the chemical name(s) and concentration, time from exposure, mode of exposure (i.e., splash, projectile, and propellant), the presence of eyewear, and vision status prior to injury.

Beyond isolated exposures from accidental or occupational injuries, be aware of other symptoms particularly cough, wheezing or respiratory difficulties, nausea and vomiting, dizziness, muscle weakness, or fasciculation that may represent an impending CW attack. Clearly, management of life-threatening injuries takes priority over a detailed ophthalmologic evaluation per ATLS/AHLS protocols. Maintain proper isolation and decontamination precautions with appropriate use of PPE and notify EMS or command of suspected CW agent(s).

Continue irrigation for a minimum of 15–20 minutes and administer topical anesthetics if available following the initial irrigation phase. Remove contact lenses if visually present. Runoff irrigation fluid should be kept from cross-contaminating other nonexposed areas. Allow 3–5 minutes prior to assessment or reassessment of tear film pH. This is more than enough time to conduct a brief field evaluation of the adnexa, anterior, and posterior structures of the eye.

With a pen light, the eyes and adnexa are thoroughly examined for gross contaminants, particular matter, or chemical concretions. This includes mandatory eversion of upper and lower lids. Any significant findings should prompt removal of particles or resumption of irrigation. Examine external surfaces for skin burns or tissue loss, which may lead to corneal damage from incomplete lid closure. The corneal surface and anterior chamber can be seen with the naked eye and tangential lighting. Healthy corneal epithelial cells have a reflective luster or shine. In direct examination of the iris, note any gray areas of stromal opacification or loss of clarity. Corneal opacification, epithelial defects, and limbal ischemia should be noted. The presence and amount of blanching at the corneal-conjunctival border reflects limbal ischemia and has prognostic value (see chemical injuries). Slit lamp examination with mydriatics provides a more detailed assessment of these structures, including the lens, but would not be appropriate prior to completion of irrigation decontamination. Evaluate direct and consensual pupillary light reflexes, and note any differences in baseline diameter, asymmetry, and sluggish movement. The presence of normal reflexes does not reflect the absence of retinal injury or damage to the visual cortex that lies outside this pathway.

Any suspicion of penetrating injury requires eye shielding and immediate triage to CSH or forward hospital for further management. Consider withholding mydriatics in patients with suspicion of head injury, as these agents will render pupillary reflexes nonfunctional. Avoid alpha agonists such as phenylephrine due to its vasoconstriction effects and the potential to enhance pre-existing ischemic injury [49].

Evaluate ocular movements, and note the presence of nystagmus, disconjugate movement, diplopia, and lid positions (both open relaxed and closed). In the absence of formal tonometry, a "field assessment" of IOP can be difficult [50]. Some authors mention tactile feedback by gentle fingertip pressure over a closed lid, in addition to light shadow test across the iris, but neither are essential to early assessments. Assess pH prior to evaluation of visual acuity. Continue irrigation cycles with pH reassessment not more often than once every 15–20 minutes to not interfere with the efficacy of decontamination. Irrigation endpoints include absence of gross contaminates and normalization of tear film pH (7.3–8.0).

The importance of visual acuity assessment cannot be overstated. Visual acuity is the single most important prognostic factor after eye injury. Each eye should be assessed independently. In the absence of a Snellen chart, have the patient read print at a recorded distance. Assess for gross visual field defects in all four quadrants of each eye. If print cannot be read, have the patient count fingers at progressively shortened distances until the patient can do this reliably "counts three fingers at 4 feet." If finger counting is not possible, check for detection of hand movements and, finally, localization of light perception if necessary [51]. In the absence of significant anterior chamber injury, intact visual acuity and visual fields confirm function of both the posterior bulb structures and visual cortex.

Once in a controlled setting or acute care facility, the patient may undergo a complete ophthalmologic exam, including formal dilated slit lamp examination, fluorescein staining, and direct or indirect funduscopic assessment.

The presence of significant defects or visual impairment may reflect underlying chemical injury, systemic toxicology, or CNS dysfunction and injury. Accurate assessment and recognition of injury patterns greatly impact medical outcomes and long-term disability [52]. Providers need to practice these assessment skills and decontamination protocols such that they can be performed efficiently and under potential duress.

Vision Testing

Following injury, in-depth assessment of vision can be accomplished via a variety of neuro–/electro-physiologic procedures. The most commonly employed tests are visual-evoked potentials (VEP), electroretinography (ERG), and electrooculogram (EOG).

VEPs are a form of sensory-evoked potential (SEP), which is simply an electrical signal from the CNS in response to the presentation of a stimulus. There exist various types of SEPs, including auditory-evoked potentials and somatosensory-evoked potentials (peripheral nerves), to assess competence of their respective neural pathways.

Using transducers, these electrical signals are graphically recorded as response amplitude and frequency. Repetitive signals demonstrate a constant pattern referred to as steady-state evoked potentials (SSEP). These measurements are specific to a particular stimulus (e.g., high-frequency versus low-frequency light flicker will demonstrate different amplitude response patterns). Simultaneous stimulation involves multiple SSEP recordings from different scalp locations, which allows for spatial analysis brain stimulation. Sweep technique plots response amplitudes against size of visual stimulus and frequently employs a changing checkboard pattern several times per second. This technique is useful in measuring visual development and contrast sensitivity in the first years of life [53]. In evoked potential feedback, the luminal strength of the visual stimulus is automatically adjusted to maintain constant response amplitude. Then the color or wavelength is changed, and a plot of luminance stimulus versus wavelength provides a graphical measure of spectral sensitivity of the visual system [54].

VEPs were first reported by Adrian and Matthews in 1934, who discovered changes in occipital lead electroencephalograms (EEG) under visual stimulation with light. Later, specific nomenclature was developed, and the recording of maximal amplitudes along the calcarine fissure (primary visual cortex) was discovered.

Electroretinography (ERG) measures retinal electrical responses to light stimulus with electrodes placed on or near the cornea. Light stimulus in reference to background lighting will demonstrate specific function of rods and cones (e.g., response from light stimulus in a dark room will measure rod function, whereas stimulus under daylight background will be more representative of cone function). Bright light will generate a biphasic wave with an initial negative deflection (a wave), which represents photoreceptor response. The trailing wave (b wave) reflects retinal bipolar cells and Muller cell function. From animal studies in these areas, a standard set of ERG protocols has been developed for use in the clinical assessment of human patients [55]. VEP and ERGs are valuable tools when

suspecting xenobiotic-induced retinal or macular pathology because they can demonstrate dysfunction when clinical symptoms are subtle and injury is still reversible. The EPA has published guidelines on the use of EP testing in the context of toxicological studies [56].

Electrooculograms (EOG) measure standing electrical potentials between the front (cornea) and back (retina) of the eye. Unlike ERGs, they do not measure response to temporal visual stimuli but are mainly used to measure eye movements. Metabolic activity in the RPE generates light-sensitive and light-insensitive potentials. In this setting, EOGs can be used to evaluate healthy functioning of the RPE by comparing peak resting potentials during light and dark adaptations.

Beyond the diagnostic capabilities of tertiary referral centers, the keen provider should be aware of potential xenobiotic exposures in patients experiencing trouble with color discrimination, abrupt changes in night vision, dark adaptation, or contrast differentiation. Kollner's rule is a term used in ophthalmology that pertains to the progressive nature of color vision loss that is secondary to eye disease. This rule states that outer retinal diseases and media changes result in predominant blue-yellow color defects, while diseases of the inner retina, optic nerve, and visual pathway will result in more red-green defects [57, 58].

Color and contrast changes often are the first symptoms in isolated toxin-induced retinopathies. Systemic xenobiotic exposures most likely will target the outer retina or RPE due to its proximity to the choriocapillaris blood supply. Other xenobiotics such as methanol easily penetrate the retina, CNS, and optic nerve, causing delayed toxicity via by-products of metabolism. Color vision testing has been used to rapidly screen occupational and environmentally exposed populations. A number of standard colorimetric tests exist, including the Farnsworth-Munson 100 Hue (FM-100) test and a simplified 15 chip version (Farnsworth D15) panel. Test subjects are required to arrange the color chips in a sequential chromatic order. Those individuals with color perception deficits will be unable to place in the correct order. Whether drinking water contaminated with heavy metals to methanol-adulterated alcoholic drinks, attention to the early symptoms of visual dysfunction may be nothing less than vision saving.

Increased intraocular pressure (IOP) is a frequent complication of severe chemical injury to the eye, often secondary to scarring of the trabecular meshwork and formation of posterior synechia. Vigilant reassessment of IOP via tonometry and ultrasound bio microscopy is often necessary to avoid potential secondary injuries.

Chemical Warfare Agents

Chemical warfare (CW) agents have been in use for centuries, but it was not until after the industrialization of Western Europe in the mid-nineteenth century that allowed for the mass production and accelerated development of warfare agents. In April of 1915, the devastating use of chlorine gas by German forces in Belgium marked the beginning of the modern chemical weapons era. This led to the development and use of other agents including sulfur mustards and phosgene. These agents, used by both axis and allied forces during World War I, were responsible for an estimated 100,000 deaths and excess of 1 million casualties (U.S. War Department Feb 1924, Amended Office of the Secretary of Defense on 7 Nov 1957).

The Lieber Code of 1863, which was signed by President Abraham Lincoln, provided instructions for the civilized conduct of Union soldiers during wartime. In essence, it was the first written recital of the customary laws of war and includes provisions on military jurisdiction, martial law, and the treatment of spies, deserters, and prisoners of war [59]. It also forbade the use of poisons. The Hague Conventions of 1899 and 1907 were the first multilateral treaties, which further defined the laws of war and created the first binding international court for compulsory arbitration. In the aftermath of World War I, the Geneva Protocol (1928) to the Hague Conventions banned the use of all forms of chemical and biological warfare. Despite their limited use, active research in CW agents continued. The Chemical Weapons Convention (1997)

Table 14.3 Standard Iraqi chemical agents in 1991

Name	Agent type	Comments
CS	Riot control	Low-toxicity agent for harassment and deception
Cyclosarin (GF)	Nerve	Deployed and stored in GB/GF mix
Sarin (GB)	Nerve	Deployed and stored in GB/GF mix. Some degraded Iran-Iraq war-era GB-only munitions found at Al Muthanna
Sulfur mustard (H, HD)[a]	Blister	Most prevalent agent in munitions and bulk storage containers
VX	Nerve	Quantities unknown but probably small when compared to other standard agents

[a]H is a common symbol for mustard gas. HD is variously used to designate distilled or otherwise highly pure sulfur mustard. Iraqi mustard was not distilled but was often greater than 90% pure

is an international arms control treaty with comprehensive bans on the development, production, and use of CW agents and provides destruction timelines for existing stockpiles.

Despite these international treaties, the recent use of CW agents in the Iraq-Iran War (1982–1988), Kurdistan rebellion (1988), and the Syrian Civil War (2013) underscores the ongoing need for vigilance in military training and healthcare provider education in these areas (Table 14.3).

In general, CW agents are classified according to their physiologic effects. These categories include vesicants or blister agents, nerve agents, choking or pulmonary agents, blood agents, and riot-control agents. Volatility is important to note, as this impacts whether the agent is classified as persistent or nonpersistent. Less volatile agents like sulfur mustard and VX tend to persist in a given exposure area, whereas more volatile agents like chlorine, hydrogen cyanide, and phosgene evaporate and disperse quickly.

Vesicant or Blister Agents

In chemical warfare, *sulfur mustard* (HD) or "mustard gas" is the prototypic vesicant or blister agent [67]. It can exist as an oily liquid or vapor and colorless to yellow-brown in color and sometimes smells of garlic, onion, or mustard. A vapor hazard only above body temperatures, it is heavier than air and typically accumulates in low-lying areas. In cold climates, it may persist in these areas for several days. It is more effective in warmer climates and in higher humidity weather but will usually dissipate within 24–48 hours. At temperatures above 300 F, decomposition occurs with the release of toxic fumes including sulfur oxide and hydrochloric acid [60]. It causes severe delayed burns to the skin, eyes, and respiratory tract. Sulfur mustard can be absorbed into the body by inhalation, ingestion, and dermal contact. The median incapacitating dose is 100–200 mg-min/m^3 and results in moderate-to-severe injuries [61]. Death is typically via inhalation injury.

Sulfur mustard is lipophilic and rapidly penetrates dermal surfaces including the cornea, often resulting in severe keratoconjunctivitis. Skin irritation, pain, and blistering are often delayed for several hours. The severity of symptoms depends on environmental conditions and the degree of exposure. Ocular findings include lacrimation, painful blepharospasm, and severe corneal and conjunctival edema similar to UV keratitis. The use of sulfur mustard during the Iraq-Iran War demonstrated that even with mild exposures, up to 90% of exposed experienced visual disability and were unable to return to combat duty for nearly 1 week [62, 63].

Sulfur mustard is a highly reactive alkylating agent that reacts with sulfhydryl, carbonyl, and amino groups. This causes denaturing of cellular proteins and inhibits DNA replication [64, 65]. In severe cases, pulmonary injury is complicated by septicemia and bone marrow suppression. Similar to chemical burns, significant exposures result in corneal defects and limbal ischemia on exam. IOP may be elevated. Severe cases result in prolonged keratitis, with recurrent corneal ulceration, scarring, and impaired visual function.

Depending on the severity of exposure, the majority of individuals with corneal injuries heal without sequela. Moderate or severe exposures can develop a chronic keratopathy, which can be delayed for several years [66].

Traditional surgical interventions have had mixed results, and the benefits from anti-inflammatory medications are temporary. This suggests an ongoing underlying mechanism to this condition [73]. Animal studies have shown an injury-dose response to sulfur mustard with corneal endothelial cell injury, abnormal morphology, epithelial bullae, and thickened Descemet membrane [68]. These findings suggest ongoing endothelial failure as a potential mechanism for chronic anterior keratopathies.

Other studies have attempted to characterize the temporal development of delayed keratopathy following sulfur mustard exposure. Chronically injured corneas were noted to have increased matrix metalloproteinase (MMP) activity, poor re-innervation, and limbal damage consistent with limbal epithelial stem cell deficiency (LSCD) [69]. Investigation of anti-inflammatory agents (NSAIDS), MMP inhibitors (doxycycline), neurotropic factors, and amniotic membrane transplantation have shown variable benefit. These interventions blunt the acute injury phase and delay the onset of chronic injury patterns. Further research is needed to better elucidate these mechanisms and to better match patients with specific interventions.

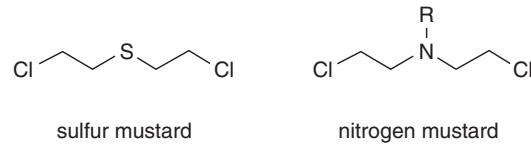
Limbal stem cell transplantation is effective in select patients. Unfortunately, severe injuries (Dua V–VI) associated with near-total limbal stem cell loss have variable success, even in the setting of aggressive surgical interventions. Traditional limbus tissue grafts are heterogeneous cell populations, which contain variable amounts of limbal stem cells (LSC). Recent work with autologous limbal cell grafts has yielded promising results for the treatment of LSCD [70]. Frank recently discovered ABCB5 as a novel molecular marker for mammalian LSC. ABCB5 monoclonal antibody-based purification yielded purified LSC grafts that were able to fully restore the cornea upon grafting to LSC-deficient mice [71].

The use of N-acetyl L-cysteine (NAC) to increase glutathione levels may provide benefit in preventing or reducing toxicity related to exposure to chemical irritants (particularly sulfur mustard) [72–75].

Treatment includes appropriate triage and immediate decontamination, which may be difficult due to significant latency with onset of symptoms. Sulfur mustard may persist on clothing for several days. After removal of clothing, exposed areas should be dusted with Fuller's earth or reactive skin decontaminant lotion (RSDL), followed by soap and copious water irrigation [39]. Ocular irrigation with buffered solutions, such as DRS, if available, otherwise use any neutral solution available (e.g., water, milk, and iced tea; see *decontamination*).

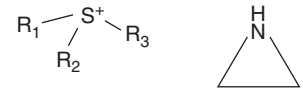
Although there are no specific antidotes for systemic mustard toxicity, there are several interventions under investigation, including N-acetyl cysteine (NAC), sodium thiosulfate [76], and amifostine [77].

Like sulfur mustard, *nitrogen mustards* are powerful blister agents with cytotoxic effects. Nitrogen mustard gas (HN2) was stockpiled by several nations during World War II, but it was never used in combat. Production and use is strongly restricted as are all schedule 1 substances within the Chemical Weapons Convention (Organization for the Prohibition of Chemical Weapons; http://www.opcw.org).



sulfur mustard nitrogen mustard

Nitrogen mustards have the same toxicity as sulfur mustards. Rapid elimination of chloride group(s) leads to the formation of a highly reactive aziridine (nitrogen) or sulfonium (sulfur) cyclic group that readily causes alkylating injury to DNA.



Sulfonium Aziridine

Due to their known cytotoxic effects, nitrogen mustards were studied for the treatment

of lymphomas at Yale in 1942. These studies demonstrated how nitrogen mustard caused temporary regression of lymphomas in mice. It was also found that reactivity of the ethyleneimmonium ring with thiosulfate provided a potential antidote to nitrogen mustard's cytotoxic effects [72]. Thus began the modern age of chemotherapy.

Several related chemotherapeutic agents have since been developed, including ifosfamide, cyclophosphamide, chlorambucil, and melphalan. The original nitrogen mustard (Bis-2-chloroethyl methylamine or mustine) is no longer in use due to its toxicity.

Lewisite (agent L) (Fig. 14.4) was first synthesized in 1904 by Julius Arthur Nieuwland and later purified by US chemist Winford Lee Lewis (1919). Lewisite (dichloro[2-chlorovinyl] arsine) is an organoarsenic compound. In its pure form, it is colorless and odorless, (like mustards). Impurities in mass production frequently yield a yellow-brown oily liquid (similar to mustard), and a smell described as freshly cut grass or geraniums.

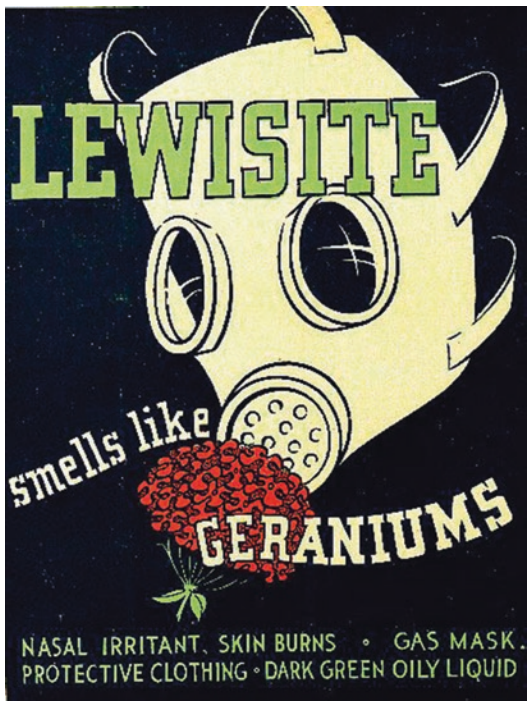
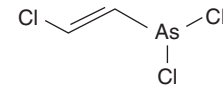


Fig. 14.4 Lewisite (agent L). Lewisite, World War II Gas Identification Posters, ca. 1941–1945, U.S. Army, National Museum of Health and Medicine, http://nmhm.washingtondc.museum/collections/archives/agalleries/ww2/lewisite.jpg



Lewisite

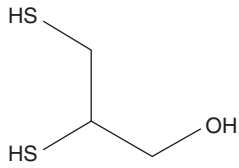
Lewisite is heavier than mustard and poorly soluble in water. Lewisite may be mixed with sulfur mustard to lower the freezing point of sulfur mustard and increase its effectiveness at lower temperatures [78]. In addition to its potent vesicant effects causing severe pulmonary injury, it also has systemic effects including refractory hypotension and hepatic injury. Cellular toxicity is secondary to the reaction of arsenite ions with thiol groups of biologically active proteins involved in energy (ATP) production [79].

Unlike mustard agents, the irritant effects of lewisite are immediate. Exposure causes rapid onset of eye and skin irritant effects followed by pulmonary injury and finally systemic toxicity. Lewisite is highly lipid soluble and deeply penetrates dermal tissues. Toxicity of lewisite is many times than that of mustard agents but due to the rapid onset of irritant effects at relatively low concentrations, most exposed individuals take immediate protective action, limiting further contact [80].

Ocular effects include pain, blepharospasm, edema, and chemosis within the first minute of exposure. Severity of corneal injury is concentration (dose) dependent. Corneal irritation occurs at concentrations as low as 0.1 mg-min/m^3, whereas injury occurs at doses nearly ten times higher. Permanent ocular damage occurs at 15 mg-min/m^3, which is nearly 1 minute at olfactory (geraniums) detection (14 mg min/m^3) threshold [80].

Compared to mustard agents, lewisite penetrates tissues with greater efficiency. Miosis and uveitis are demonstrated shortly following higher dose exposures, suggesting rapid corneal penetration, with injury to the corneal endothelial cells (CEC) and possibly posterior eye structures as well [81]. Although effective treatment is time critical, lewisite-induced injury can be mediated

by the administration of 2,3-dimercaptopropanol (BAL). BAL is a useful chelator for many heavy metals and has multiple thiol groups that form stable bonds with arsenic in lewisite.



2,3-dimercaptopropanol (BAL)

BAL itself is toxic with a narrow therapeutic range. Most patients receiving therapeutic doses of BAL intramuscularly will experience a transient increase in pulse and blood pressure.

Unfortunately, BAL is poorly water soluble, and no specific ocular formulation exists. In the rabbit cornea model, a 5% BAL compounded topical ointment or solution applied within 2 minutes of lewisite exposure prevented the development of a significant reaction [82]. Less toxic derivatives DMSA (meso-2,3-dimercaptosuccinic acid) and DMPS (d,l-2,3-dimercapto-1-propanesulfonic acid) are available for oral or parenteral administration. These newer agents and their chelates are hydrophilic, and they do not redistribute the toxic metals into the brain [83]. The toxicity of these antidotes is relatively low and include mild neutropenia and transaminitis [84].

Regardless of specific vesicant exposure, aggressive use of decontamination solutions, anti-inflammatory agents, and aforementioned immune modulators are necessary to help minimize the development of chronic inflammation, delayed keratopathy, and recurrent corneal ulceration. These interventions will also translate into better surgical outcomes for those patients requiring delayed keratoplasty, corneal transplantation, or limbal stem cell transplantation.

Choking Agents

First used in WWI, *chlorine gas* is a powerful pulmonary irritant or choking agent. Airway and pulmonary effects from CW agents are largely related to dose or length of exposure, which is affected by water solubility. Highly water-soluble agents cause immediate mucosal and upper airway irritation, allowing victims to take protective action, potentially limiting further injury. Poorly water-soluble agents like phosgene often cause delayed pulmonary toxicity from prolonged exposure times. Chlorine has intermediate water solubility and thus has the capacity to cause both upper airway irritation as well as pulmonary injury from prolonged low concentration exposures.

Significant exposures rapidly result in dermal, eye, and pulmonary irritant effects with morbidity largely due to pulmonary injury. Like mustards, it is heavier than air and may collect in low-lying areas. Chlorine is highly corrosive, and its toxic effects result from reaction with water to form hypochlorous and hydrochloric acid. In addition to causing direct cellular injury, these by-products lead to the production of free radicals that cause further damage to cellular proteins, pulmonary surfactant, and enzyme systems [85–87].

Clinical effects are dose dependent with mild irritation of mucosal membranes at 1–3 parts per million (PPM), onset of pulmonary effects at 15–20 ppm, and fatalities over 200 ppm [88]. Severe pulmonary exposure results in pulmonary edema and ARDS (acute respiratory distress syndrome). Dermal contact results in irritation and potential blister formation. Ocular effects include chemosis and conjunctivitis, with higher concentrations rapidly causing corneal epithelial defects [88].

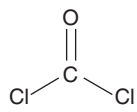
Unlike most vesicants, chlorine-related casualties do not represent a significant contamination risk to rescue workers. Standard decontamination should include removal of clothing and copious irrigation for any dermal or eye symptoms. There is no specific antidote for chlorine exposures. Following decontamination, mainstay treatments include parenteral steroids and inhaled beta-agonists for respiratory complications [89, 90]. A small case series demonstrated benefit with inhaled sodium bicarbonate (3.75%) nebulizer without adverse effects [91].

Chlorine has many uses, ranging from household cleaning agent and disinfectant to water

treatment and bleaching agents of wood pulp in pulp mills. Annual US industrial production exceeds 10 million metric tons (US Census Bureau 2009). Most US production is limited to less than a dozen states, requiring regular transport of large quantities to metropolitan areas [92].

Fatalities have resulted from both domestic accidents and terrorist attacks abroad. In January of 2005, a railroad accident in Graniteville, SC led to the release of nearly 60 tons of chlorine gas, resulting in nine fatalities and over 500 visits to local emergency departments for related symptoms [93]. Between January and June of 2007, insurgents in Iraq conducted 15 separate attacks using improvised (usually tanker truck) chlorine bombs that were responsible for over 100 fatalities and countless injured [94]. Clearly, this agent remains a viable threat for terrorist attacks both here in the United States and abroad. Although case fatality rates for those surviving to hospital evaluation are low, the sheer number of potential patients seeking medical care may rapidly overwhelm local and regional medical resources.

Carbonyl dichloride or *phosgene* ($COCl_2$) is a colorless gas well known for its use as a chemical weapon during World War I. Like chlorine, phosgene is heavier than air and will collect in low-lying terrain. It is used today as a chemical reagent in the production of other organic compounds and pharmaceuticals. The majority of phosgene is used in the production of isocyanates (e.g., toluene diisocyanate and methylene diphenyl diisocyanate), which are used in the production of polyurethanes. Toxicity from exposure to methyl isocyanate is clinically indistinguishable to that of phosgene. Because of these large-scale industrial uses, it is classified as a schedule 3 chemical under the Chemical Weapons Convention [2].



phosgene

Unlike chlorine gas, phosgene (Fig. 14.5) is poorly water soluble and therefore reaches the pulmonary alveolus without significant upper airway symptoms or irritant warning. Phosgene is a highly reactive acylator, reacting with nucleophilic moieties at its site of predominant contact (alveolus) [95]. Clinical effects include cough, dyspnea, hypotension, vomiting, and dermal irritation [96]. Death is secondary to pulmonary edema and acute lung injury (ALI). Phosgene was responsible for the majority of chemical deaths during WWI. It is not clear whether phosgene-induced ALI is secondary to cardiogenic dysregulation or direct pulmonary injury. Histopathology of the lungs reveals exposure-dependent edema and a progressive bronchiolar inflammatory response, with infiltration of polymorphonuclear cells and lymphocytes [97]. Recent animal studies seem to favor cardiogenic dysfunction and demonstrated the inefficacy of anti-inflammatory agents post exposure [98–100]. Some authors recommend that NAC and bronchodilators should be given in all cases that

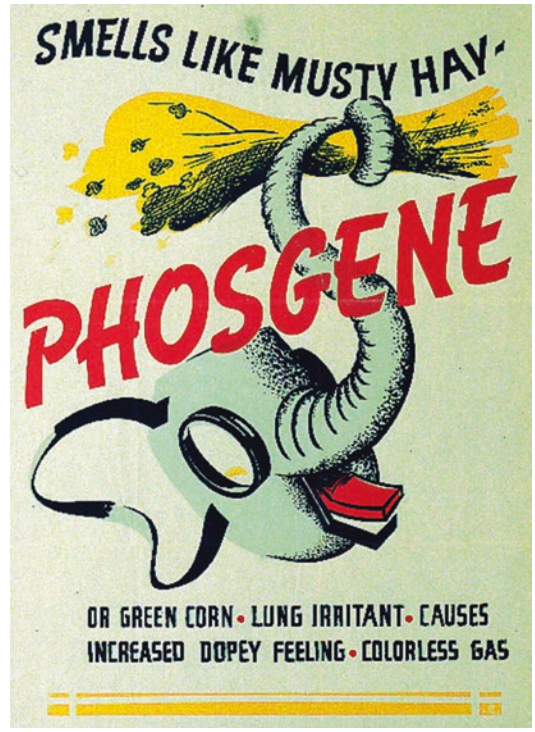


Fig. 14.5 Phosgene. Phosgene, World War II Gas Identification Posters, ca. 1941–1945, U.S. Army, National Museum of Health and Medicine, http://www.medicalmuseum.mil/assets/images/galleries/world_war_II/phosgene.jpg

have respiratory symptoms, with an additional benefit potentially from corticosteroids [101].

Olfactory detection described as "freshly mown hay" or "cut green corn" occurs at 1–2 ppm but without immediate symptoms. Toxicity of phosgene is enhanced by its lack of water solubility. Unlike chlorine, low-level gas concentrations may go unnoticed and result in significant morbidity due to a higher cumulative exposure. The same 1–2 ppm exposure after several hours of latency leads to significant dermal irritation and pulmonary edema [102]. The lethal dose of phosgene in humans is nearly 500 ppm/min. Exposure to 5 ppm for 100 minutes is equally as fatal as exposure to 50 ppm for 10 minutes [103].

Although pulmonary injury dominates medical management with significant exposures, ocular symptoms in these cases often include lachrymation and conjunctivitis. Corneal epithelial defects may result in delayed keratopathy, but permanent eye injuries are rarely reported in the literature. Unlike lewisite, phosgene's irritating quality can be mild and delayed, which may result in exposure for prolonged periods [104]. Dose-dependent exposure determines time of symptom onset and degree of injury. Unfortunately, this usually results in significant exposure by the time of moderate symptom onset [105].

This has led to the development of chemical sensors, which combine with phosgene to initiate intramolecular cyclization and convert nonfluorescent molecules to highly fluorescent products with detection limits as low as 1 nM [106]. Residual vapor detection (RVD) kits, which utilize silica impregnated with colorimetric chemicals, are also effective for phosgene detection, in addition to several other CW agents.

Nerve Agents

Nerve agents are phosphorus-containing organic chemicals or *organophosphates* (OP) that bind to acetylcholinesterase, a key enzyme that regulates neurotransmission by metabolizing acetylcholine within the synapse.

Originally developed as an insecticide, *tabun* (GA) was first synthesized in 1936 by German scientist Dr. Gerhard Schrader. Recognizing the extreme toxicity of this agent, other agents were developed under the Nazi regime including *sarin* (GB) in 1938 and *soman* (GD) in 1944. Cyclosarin (GF) was later developed in 1949. These agents, known as "G-series" nerve agents because of the initial development by German scientists who first synthesized them, are classified as weapons of mass destruction by the United Nations.

After WWII, further development of these agents led to the discovery of even more toxic pesticides. Diethyl S-[2-(diethylamino)ethyl] phosphorothioate (Amiton) was marketed as a highly effective pesticide by Imperial Chemical Industries, a British chemical company. It was later withdrawn from commercial use due to its extreme toxicity but not before recognition by the British government. This led to the development of a new class of nerve agents known as "V-series" agents. Unlike G series agents, V agents are persistent with a consistency similar to oil, making cross-contamination to health care providers problematic.

Further development of insecticides with strict regulatory guidance has led to drastically improved public safety. Created by the United Nations, an international classification system (Globally Harmonized System of Classification and Labeling of Chemicals or GHS) was developed to assign consistent criteria for classification and labeling at a global level (Globally Harmonized System of Classification and Labelling of Chemical (GHS) -2nd ed. United Nations, 2012).

OP toxicity is secondary to inhibition of acetylcholinesterase (AChE) causing accumulation of excess acetylcholine neurotransmitter in the nicotinic and muscarinic synapses. Excess stimulation of these receptors leads to a cholinergic toxidrome (see Fig. 14.6). *Muscarinic* receptor overstimulation causes miosis, lachrymation, salivation, diarrhea, loss of bladder control, and potentially lethal bradycardia, bronchorrhea, and bronchoconstriction (aka "killer B's"). Excess *nicotinic* effects include transient *mydriasis*, tachycardia, muscle spasm, and seizures, progressing to paralysis and coma. Weakness often starts peripherally and progresses to respiratory muscle paralysis with both early (<4 hours) and late (>24 hours) respiratory failure [107].

Cholinergic toxidrome	
Nicotinic	Muscarinic
Muscle cramps	Diarrhea
Tachycardia	Urination
Weakness	Miosis
Twitching	Bradycardia, bronchospam, bronchorrhea
Fasiculations	Emesis
	Lacrimation
	Lethargy
	Salivation, seizures

Fig. 14.6 Cholinergic Toxidrome. (Modified from Cholinesterase Inhibitors, ATSDR, Oct 2007, http://www.atsdr.cdc.gov/csem/csem.asp?csem=11&po=5)

Symptom onset may be delayed with highly lipophilic compounds such as chlorpyrifos and diazinon [108].

Clinically, these agents do not cause ocular toxicity per se but are important to mention because of their ocular effects including miosis, decreased vision, and eye pain. The Tokyo subway sarin attack on 20 March 1995 left 12 dead and over 5000 people complaining of acute illness. Of the 627 patients who presented to local emergency departments, over 90% displayed miosis, and nearly half complained of decreased or blurred vision and eye pain [109]. Unlike G agents, exposure to VX may cause little or no initial ocular symptoms. Initial symptoms may also be affected by route of exposure (e.g., inhalation of G agents versus more common dermal absorption of VX) (Fig. 14.7).

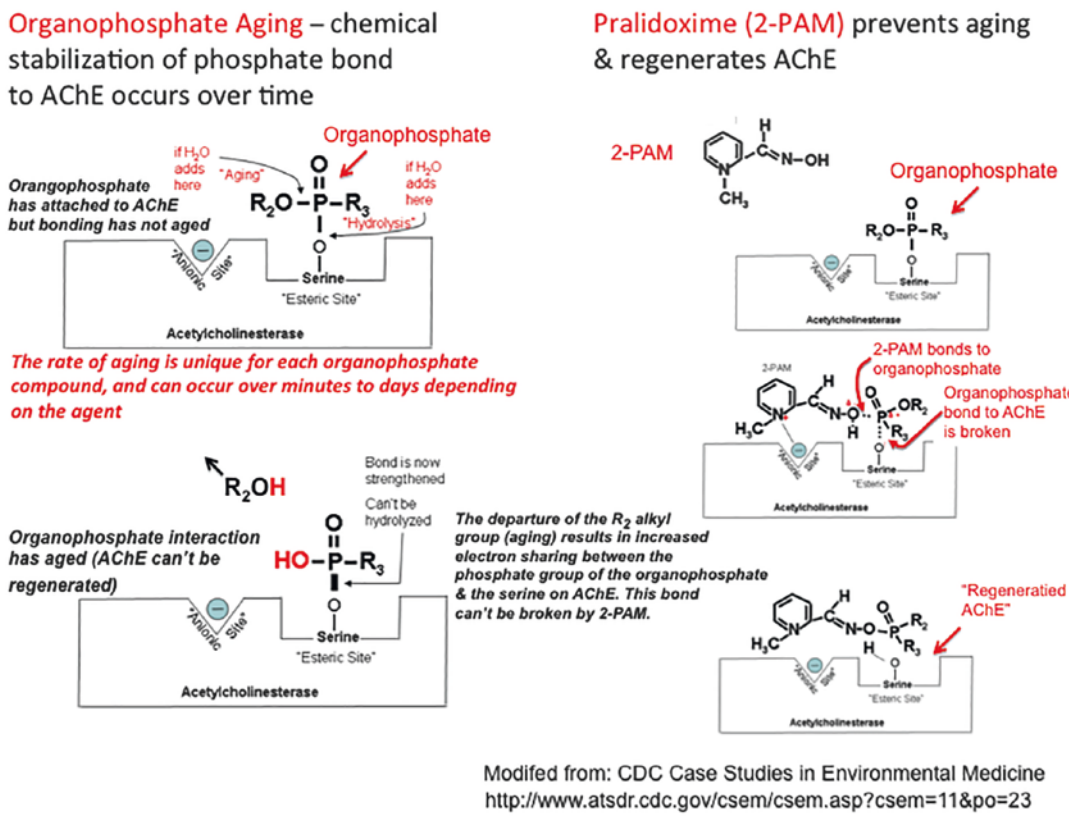


Fig. 14.7 Organophosphate aging and pralidoxime (2-PAM). (Modified from CDG Case Studies in Environmental Medicine. http://www.atsdr.cdc.gov/csem/csem.asp?csem=11&po=23)

Table 14.4 Irreversible inhibition or aging associated with high mortality rates and timing varies between different agents

Agent	LCt50 (lethal concentration mg-min/m^3)	Aging half-life
Tabun (GA)	400	<40 h
Sarin (GB)	100	<12 h
Soman (GD)	50	minutes
VX	10	<55 h

Adapted from Ivarsson et al. [202]

Inhibited (or phosphonylated) AChE may spontaneously reactivate, and they reactivate more quickly in the presence of an oxime drug (e.g., 2 PAM), or become irreversibly bound to the OP, a process known as "aging" (involves dealkylation, dearylation, and deamidation). Irreversible inhibition or aging is associated with high mortality rates, and timing varies between different agents. Soman is perhaps the one of the most potent nerve agents with an average aging time of less than 5 minutes (Table 14.4) [110].

The focus of treatment is prudent use of atropine to avoid morbidity from the muscarinic "killer B's" and pralidoxime to regenerate AChE prior to potential aging. This is in concert with thorough decontamination to avoid ongoing exposure to both patient and healthcare providers.

The adult atropine dosing is 1–3 mg intravenously (child: 0.05 mg/kg IV), with repeated doubling of dosage every 3–5 minutes as needed until drying of pulmonary secretions [111]. Children may be more vulnerable than adults with more pronounced CNS effects, and they have a larger surface area to body size, faster minute ventilation, and faster metabolic rates [112, 113]. Once control of secretions (clear lungs) is achieved, maintain with an infusion of 10–20% of the loading dose every hour, monitoring for evidence of atropine toxicity (e.g., delirium and hyperthermia) and titrate accordingly. Large doses are often required and may be needed for days depending on severity.

Treat nicotinic symptoms (fasciculations, muscle weakness, respiratory depression, coma, and seizures) with pralidoxime in addition to atropine. This is most effective if given within 24 hours of exposure but is dependent on the specific agents. Not all agents undergo aging. Administer for 24 hours after cholinergic manifestations have resolved. WHO recommendations include an initial bolus of least 30 mg/kg followed by an infusion of more than 8 mg/kg/h or 1–2 g of pralidoxime infused over 15 minutes (child: 50 mg/kg max 2 g/dose) [114]. This is followed by an infusion of 500 mg/h (child: 20 mg/kg/h max 500 mg/h). Avoid depolarizing neuromuscular agents such as succinylcholine for rapid sequence intubation, as they might have prolonged action times.

Incapacitating Agents

3-Quinuclidinyl benzilate (BZ) or "buzz" is an anticholinergic glycolate similar to atropine and scopolamine. These agents antagonize muscarinic cholinergic receptors in both the peripheral and central nervous systems. BZ was discovered by a Swiss pharmaceutical company in the 1950s, looking to develop an antispasmodic agent for gastrointestinal complaints [115]. Its development as a military weapon was highly prioritized shortly thereafter. Over 50 tons were produced for the US Army between 1963 and 1964 ("A Plan to Destroy an Old Weapon", Chemical Weekly, 1982;13–14). Aerosolized dissemination was done using thermal generators of either 3 × 50-lb canisters (M16) or a 750-lb bomb cluster (M43) [116].

BZ is odorless, nonirritating, and persistent. The ICt50 (incapacitating concentration in 50% of exposed individuals) would vary based on activity, but for mild exertion, it is approximately 100 mg-min/m^3 [117]. With respect to other hal-

lucinogens like weaponized LSD, BZ has a superior safety index, with approximately a 100-fold difference between LCt50 and ICt50. Inhalational exposure leads to fluctuating delirium with behavioral lability, dry flushed skin, and obvious mydriasis (dilated pupils). Of note, mydriasis from anticholinergic toxicity can usually be differentiated from sympathomimetic agents (e.g., amphetamines and cocaine), as the latter will constrict slightly with light reflex testing. This is not possible with anticholinergics because the pupillary constriction mechanism relies on cholinergic function and is effectively "paralyzed" under these conditions. Other symptoms include drying of secretions and constipation (opposite to nerve agent effects). Use of the Mark 1 antidote kit (atropine) could worsen symptoms, but CNS effects would most likely only be manifest after multiple doses.

After much testing, the use of BZ was eventually abandoned by the US military due to several reasons. Live dispersal would manifest as a white cloud obvious to targeted personnel. Also, the rate and duration of action are highly variable, with only half of effected individuals showing symptoms at 5 hours. Unlike nerve agents, effective inhalational delivery could be largely subverted by breathing through several layers of folded clothing, and BZ was relatively expensive to produce. Designed to be used in very specific scenarios as a means for nonlethal incapacitation, the majority of those affected required restraint to prevent self-injury with duration of action of more than 36 hours [117].

Specific antidote therapy is aimed at increasing levels of postsynaptic acetylcholine. The antidote of choice is carbamate anticholinesterase physostigmine, which, unlike pyridostigmine, is able to penetrate the CNS and reverse the central, as well as peripheral neuronal effects of BZ. Adult dosing is 1–2 mg IM or slow IV push (<1 mg/min). Rapid administration may lead to dangerous bradycardia and arrhythmias. Administration of physostigmine is clinically more effective if given beyond 4 hours following BZ exposure [118]. The successful use of antidote does not shorten the clinical course of BZ exposure (upwards of 96 hours), and given the short duration of physostigmine (60 minutes), frequent re-dosing is required (start at 2 mg/h and titrate to effect).

Although the United States considered BZ obsolete in 1977, the Soviets and Iraq were reported to continue development of BZ known as "agent 78" and "agent 15," respectively. Recent alleged reports of use of these agents include the Syria and Gouta attacks.

Fentanyl is a powerful synthetic opioid, which has been developed for use as an incapacitating agent by both the United States and former Soviet Union. In weaponized aerosol form, primary clinical effects include pin-point pupils (miosis), CNS depression, and respiratory depression within seconds to minutes of exposure. Fentanyl is approximately 100 times more potent than morphine and heroin. Ultrapotent analogs include alfentanil, sulfentanil, and carfentanil. Much controversy surrounds the suspect use of this or related agents (3-methyl fentanyl or kolokol-1) by Russian Spetsnaz in the 2002 Moscow theater hostage crisis. Over 130 of 850 hostages died from exposure to an unconfirmed chemical agent that was introduced through the theater's ventilation system prior to breach. There are unconfirmed reports that a handful of hostages were revived by the use of naloxone by EMS personnel. Fentanyl can be absorbed via inhalation, ingestion, or dermal contact.

Naloxone is an opioid receptor antagonist and can reverse the effects of fentanyl within 1–3 minutes following intravenous administration (0.4–2 mg doses). Maximum effect is within 5 minutes and the duration of action is nearly 1 hour. Doses may be repeated until desired effect.

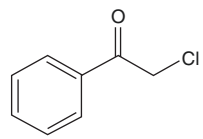
Following animal testing, the United States abandoned further development of aerosolized fentanyl (including related analogs) as an effective incapacitating agent primarily due to its unacceptably low safety index.

Riot Control Agents

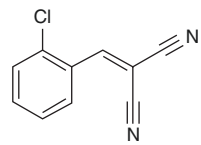
Riot control agents (RCAs) are designed for immediate incapacitation of individuals while minimizing harmful or permanent effects.

Historically, these agents have been used for centuries (e.g., Japanese use of pepper dust blown in the face of enemies). RCAs are considered harassing agents and nonlethal and designed to produce temporary disability. Most of these agents are solids at room temperature, soluble in organic solvents, and typically dispersed as aerosols. They all cause rapid onset of lachrymation, eye pain, and ocular and dermal irritation. The United States excludes these agents from the Geneva Convention on chemical weapons; however, their use during wartime is restricted to defensive use and only with written approval from the President (Gerald Ford 8 Apr 1975, Executive Order 11850 "Renunciation of Certain Uses in War of Chemical Herbicides and Riot Control Agents", National Archives; Joseph Benkert 27 Sept 2006, "U.S. Policy and Practice with Respect to the Use of Riot Control Agents by the U.S. Armed Forces", Senate Committee on Armed Services).

Chloroacetophenone (CN) was invented by German chemist Carl Graebe in the later part of the nineteenth century. It was later developed by the British and United States during WWI as a very stable and very effective lachrymator. Post WWI, it became the primary RCA of choice until gradual replacement by chlorobenzylidene malononitrile (CS) in the 1950s. Both agents act through alkylation of intracellular sulfhydryl groups on enzymatic processes. Injury is limited due to rapid regeneration of these enzymes and breakdown of the parent compounds.



chloroacetophenone (CN)



chlorobenzylidene malononitrile (CS)

Clinical effects include intense eye pain and rapid onset of lacrimation, conjunctivitis, copious rhinorrhea, salivation, pharyngeal irritation, cough, and dyspnea. These agents have excellent safety profiles. Animal studies demonstrate CN inhalation median lethal concentration-times (LCt50) between 7000 and 9000 mg-min/m^3. At these levels, autopsy revealed pulmonary edema, alveolar hemorrhage, tracheitis, and bronchopneumonia [119].

Rare deaths have been associated with indiscriminate use of these agents. A single CN grenade (128 g) was thrown into a room (27 m^3) where an assailant had barricaded himself from police [120]. He remained in the room for approximately 30 minutes, after which he was found comatose and in pulmonary edema. He was rapidly transported to a local hospital and died 12 hours later. Estimated exposure was more than 140,000 mg-min/m^3, which is approximately ten times estimated LCt50 for humans. A handful of similar case reports all involve individuals confined to relatively small, enclosed spaces. High levels of CN can cause corneal and conjunctival injury with loss of corneal epithelium [16]. Other considerations include proximity and injury from the dispersal device itself.

No permanent eye injuries have been described with exposure to the recommended harassing or "field concentration" doses of CN. Sensitization to prior exposures is a theoretical concern and has been reported in animal studies with CN and other RCAs [121].

O-chlorobenzylidene malononitrile (CS), also 2-chlorobenzalmalononitrile, is the key component in tear gas and the most widely used RCA in US law enforcement and military operations today. CS has a half-life of less than 15 minutes, making it ideal as a temporary incapacitant. The US military developed variants of CS including CS1, CS2, and CSX. These agents have increased shelf life, resist degradation, and can be used over water terrain.

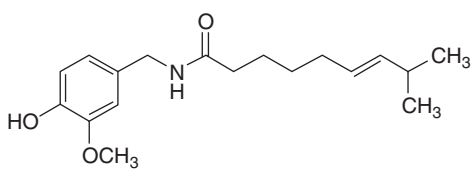
Clinical effects are similar, although CS is superior to CN as a more potent lachrymator with reduced toxicity. Animal studies on CS inhalational toxicity demonstrate LCt50 (mg-min/m^3) between 35,000 (guinea pig) and 70,000 (rat) [119, 122]. Following exposure, symptoms include immediate conjunctivitis with burning pain, blepharospasm, and profuse lacrimation with photophobia. Conjunctivitis and erythema of the eyelids may persist for an hour. CS is much less likely than CN to produce long-term ocular effects including corneal scarring or delayed keratopathy [123, 124]. Animal studies of direct

ocular exposure to CS solutions did not produce permanent damage, and all tissues were normal within 1 week [125].

Exposure symptoms include burning or stinging sensation in the nose, mouth, and throat with excessive rhinorrhea and salivation. Some individual experience pronounced coughing, dyspnea, tightness of the chest, skin irritation, and vomiting. These effects usually resolve within 10–15 minutes after cessation of exposure with the exception of ocular irritation, which usually last up to 1 hour. CS is less tolerated by individuals under exertion or in higher ambient temperatures [126].

CS is commonly used to simulate CW agents in training scenarios for military and law enforcement (LE). Heated dispersion of CS canisters is frequently employed for mask confidence training; however, within enclosed spaces, this was found to produce several semi-volatile air contaminants [127]. The metabolic effects of CS exposure and its metabolites are well known. Current studies are looking at the products of pyrolysis from the heated dispersion of CS. GC/MS of decomposition products demonstrates a loss of cyanide from the CS molecule, and air samplings reveal the presence of both HCl and HCN in high-temperature CS dispersion [128]. Currently, the use of CS capsules is the only approved method for *enclosed-space* mask confidence training.

Oleoresin Capsicum (OC) or "pepper spray" is a mixture of pepper plant extracts (paprika, chili peppers, and jalapeno) from the *Capsicum* genus, including *Capsicum annuum* and *Capsicum frutescens*. The active ingredients in pepper sprays are capsaicinoids (vanilloid family) and include several different individual chemicals (capsaicin, norhydrocapsaicin, dihydrocapsaicin, and homocapsaicin).



capsaicin (OC)

OC concentrations may vary from 0.1% to 3% of CRC (Capsaicinoid Related Content), but manufacturers do not specify amounts of individual capsaicinoids; thus, it is difficult to make comparisons of relative strengths with different brands. Most LE agencies use a CRC between 1% and 2%, and the EPA mandates pepper sprays marketed as "bear deterrents" must contain at least 1% but not more than 2% CRC [129]. Another measure, the Scoville scale is an empirical measurement of spicy foods as reported in Scoville heat units (SHU). Testing involves exact dry weight measurements of extracted capsaicinoids sampled by trained tasters to detect the heat in a dilution sample, which is rated in multiples of 100 SHU [130, 131]. Pepper spray products demonstrate variability in the capsaicinoid concentrations, between different manufacturers, as well as from different product lots of the same manufacturer, and are not standardized for capsaicinoid content even though they may be classified by SHU [132].

Synthetic capsaicin analogues, such as nonivamide (pelargonic acid vanillylamide or PAVA), has similar effects and is more heat stable than capsaicin [133, 134]. In addition to its use as a RCA, nonivamide is used in food flavorings and spice blends, as well as a cheaper pharmaceutical alternative to capsaicin in therapeutic liniments.

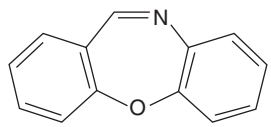
OC is widely used by the US government and LE agencies. Like CS and CN, it is a highly effective dermal and upper respiratory irritant. Primary physiologic effects include intense ocular and dermal irritation with pain, blepharospasm, and excessive lachrymation. Ocular exposure causes involuntary closure of the eyes and temporary visual impairment for approximately 15 minutes. Visual acuity normally returns within 5–10 minutes following decontamination, although individuals frequently experience pain for up to 30 minutes [135]. The LD50 of OC is route dependent. Human LD50 for dermal exposure is estimated at 500 mg/kg, whereas oral is approximately 200 mg/kg, inhaled is 2 mg/kg, and intravenous is 0.5 mg/kg [136]. Rats fed capsaicin 50 mg/kg per day for 60 days developed no significant untoward effects [137, 138].

Physiologic effects are secondary to capsaicin binding to sensory receptors TRPV1 (transient receptor potential vanilloid receptor 1). TRPV1

functions in the detection and regulation of body temperature, as well as provides sensation of thermal pain (nociception). Capsaicin-induced bronchospasm, mucosal edema, and neurogenic inflammation are mediated by TRPV1, increased release of substance P and neurokinin A [139, 140].

Dosage varies based on delivery device, but the most commonly used carrier is isopropyl alcohol [141]. This may complicate ocular injuries when used at close range. Although OC use in LE is common and generally considered very safe, direct exposure to high concentrations can lead to permanent injury. Rare human case reports site epithelial necrosis, limbal ischemia, and ultimate permanent peripheral field defects due to conjunctivalization following direct spray exposure to OC [142]. Animal studies have shown keratitis and neurotrophic injury following systemic exposure to capsaicin [143]. Children may be particularly sensitive to long-term injury. A case report of OC exposure in a toddler demonstrated conjunctival proliferation and scarring at the limbus despite topical corticosteroid treatment [144]. It is suspected that OC exposure may release neuropeptides which induce inflammation separate from traditional immune-mediated mechanisms. This results in neurogenic inflammation and loss of blink reflex, which can last for several days and increases risk of severe corneal injury [145].

Dibenzoxazepine (CR) was developed for use by the British military in the early 1960s. It is the latest of the "C" series RCAs and the most potent incapacitator of the group. Similar but more powerful than CS, CR causes immediate incapacitation with intense blepharospasm, temporary functional blindness, painful skin irritation (exacerbated in moist areas), and coughing with dyspnea [145]. Attempts at decontamination with water often worsen dermal pain and these affected areas can remain sensitized for days. Avoid cross-contamination and decontaminate with soap and water as with other "C" agents [146].



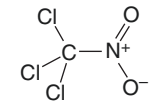
dibenzoxazepine (CR)

CR is a solid at room temperature and very stable. Dispersal is usually by particle emulsion in propylene glycol and release from heated canisters. As a vapor, CR is heavier than air and hydrolyzes slowly in water, and thus will persist in low lying areas.

Animal toxicology studies reveal that CR is less toxic than CN and CS [147]. Respiratory effects include dyspnea with diminished FEV for 20 minutes after CR inhalational exposure [148]. Animals exposed to aerosol doses of 80,000 to 160,000 mg-min/m^3 revealed alveolar hemorrhage and edema only on microscopic lung examination [149]. Intense dermatologic effects typically last for 30 minutes, followed by gradual diminution over several hours [147].

Clinical ocular effects include intense lachrymation and conjunctivitis lasting less than 1 hour. Animal studies with corneal exposures to both CR aerosol and solution (in polyethylene glycol) up to 17,000 mg-min/m^3 produced mild injection and dose-related corneal edema which cleared over several days without any permanent effects [150].

Chloropicrin (PS) or nitrochloroform was first discovered in 1848 by Scottish chemist John Stenhouse. The name chloropicrin comes from his use of a chlorinating agent applied to picric acid. Among his many discoveries, he is probably best known for his description of the absorbent properties of wood charcoal to disinfect and deodorize, which led to his invention of charcoal air-filters and charcoal respirators [151].



chloropicrin (PS)

PS is colorless, poorly water soluble, and volatile at ambient temperatures. Although used as a harassing agent in WWI, it might be better thought of as a pulmonary or choking agent. Current formulation is by the reaction of nitromethane with sodium hypochlorite, and it is used today as a highly regulated broad-spectrum fungicide and insecticide [152].

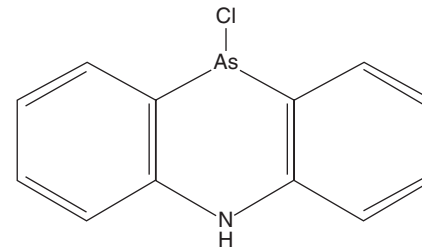
Due to its dermal and respiratory irritant effects, WWI German forces used concentrated PS gas as a tear agent against allied forces. Although not as toxic as phosgene or the vesicants of WWI, exposure would provoke vomiting, causing soldiers to remove their masks, exposing them to more CW agents.

Unlike phosgene, PS is immediately irritating to the nose, eyes, lungs, and skin at 1–2 PPM [153]. Victims report an unpleasant taste followed by nausea, vomiting, and headache. PS can also be absorbed systemically via inhalation, ingestion, and dermal contact. PS is a volatile aliphatic nitrate that causes direct cellular injury at the site of dermal contact. Systemic effects in animal studies include methemoglobinemia and modification of free sulfhydryl groups [154]. It is not clear the exact mechanisms, whether related to the parent compound or secondary metabolites, but evidence indicates oxidative damage which may be reversed by antidotes such as NAC [155, 156]. Increasing exposure leads to pulmonary edema and death via early asphyxiation or delayed chemical pneumonia [157]. The estimated LCt50 for PS is 2000 mg-min/m^3 or 300 PPM [158]. Much like the vesicants of WWI, direct skin exposure causes irritation and rash with blister formation [159].

Ocular irritation usually begins before odor threshold (<1 PPM), and includes lachrymation, blepharospasm, and conjunctivitis. These early warning symptoms usually prompt potential victims to seek protection from further exposure. Case reports of ocular exposure to liquid PS demonstrated severe corneal edema, chemosis and eventual perforation [160]. Specific animal data for ocular exposures are limited but reports demonstrate dose-dependent corneal injury patterns similar to mustard agents.

Adamsite (DM), or diphenylaminochloroarsine, was developed by German scientist Heinrich Otto Wieland in 1915, and later production was improved by Major Robert Adams at University of Illinois in 1918 [161].

DM is an odorless, yellow-green crystalline substance with low volatility (persistent) and poorly water soluble. It is usually dispersed by powder grenade or aerosolized in organic solvents. It was heavily stockpiled by the United States during WWII but not used until Vietnam. DM was often mixed with CN or CS and used by the US military during the Vietnam War as an effective means to deny enemy access of territory [162].



adamsite (DM)

Clinical effects from DM exposure typically have a latency of 5–10 minutes. Gastrointestinal effects, including nausea, vomiting, and diarrhea, predominate over dermal and respiratory irritant effects. Delayed effects had the advantage of creating a significant exposure to most victims. Primate studies demonstrated rapid onset of symptoms including rhinorrhea, salivation, vomiting, lack of coordination, and dyspnea following inhalation exposures with estimated LCt50 between 12,000 and 14,000 mg-min/m^3 [163]. These data correlate with human LCt50 estimates for exposure to highly purified DM. Human deaths have been reported following exposure to high DM concentrations within an enclosed space [164]. Postmortem examination revealed pulmonary edema and diffuse inflammation throughout the entire respiratory tract. ED50 (incapacitating dose) in human volunteers of DM ranged from 22 to 220 mg-min/m^3. Animal studies on ocular toxicity revealed dose-dependent corneal injury from temporary conjunctivitis (0.2 mg) to corneal epithelial injury and partial limbal ischemia (0.5 mg) to complete corneal opacification (1.0–5.0 mg) [164].

Classified as a malodorant, "*skunk*" is marketed as a nonlethal means for crowd control. It is commonly used by Israel Defense Forces as an improvement over the use of RCAs or rubber bullets following increasing criticism of disproportionate use of force in conflicts with Palestinian protestors.

Produced by Odortec Ltd., this product is marketed as "safe to consume", "100% eco-friendly", and "poses no health hazard" [165]. Specific chemical ingredients are lacking, though production involves the fermentation of yeast with sodium bicarbonate and production of various amino acids. The MSDS reports prolonged contact may cause dermal, eye, and respiratory irritation [166]. The product is stable and non-volatile, although its odor effects tend to persist for several days.

In contrast, natural skunk spray or skunk musk contain a mixture of several volatile sulfur-containing compounds including methylquinoline, quinolinemethanethiol, quinolinemethyl thioacetate, phenylethanethiol, butenyl disulfide, and methylbutyl disulfides identified by GC/MS [167]. The relative amounts of specific chemicals present are clearly species specific. Known to cause dermal irritation, weakness, malaise, vomiting, headache, and confusion in human exposures; cyanosis, pulmonary irritation; liver and kidney injury in animals, butanethiol or n-butyl mercaptan has threshold exposure limits set by the CDC of 0.5 ppm (27 mg-min/m^3) [168].

There are animal case reports of methemoglobinemia with Heinz body formation following exposure to natural skunk musk, later confirmed by in vitro studies [169, 170]. These studies demonstrated a dose-dependent relationship of skunk musk to the level of resultant methemoglobin, supporting a theory of oxidative stress-induced hemolysis.

These agents could represent a potential terrorist chemical threat, but due to their relative safety and very low odor detection thresholds (<0.001 ppm) (National Center for Biotechnology Information, NIH; http://pubchem.ncbi.nlm.nih.gov), their risk relative to other agents is small.

Ultraviolet Light

The toxicodynamics of many chemicals is mediated via oxidative injury, but ultraviolet radiation (UV) is the most important oxidizing agent in regard to the eye. High-energy UV-C (100–280 nm) radiation is completely filtered by our atmosphere (Fig. 14.8). We are exposed to only a small portion of UV-B (280–314 nm) and most of UV-A (315–399 nm). A typical human retina functions at wavelengths from approximately 390–700 nm [171]. Both the cornea (UV-B) and lens (UV-C) absorb UV radiation, preventing transmission of nonvisible light to posterior ocular structures and the retina.

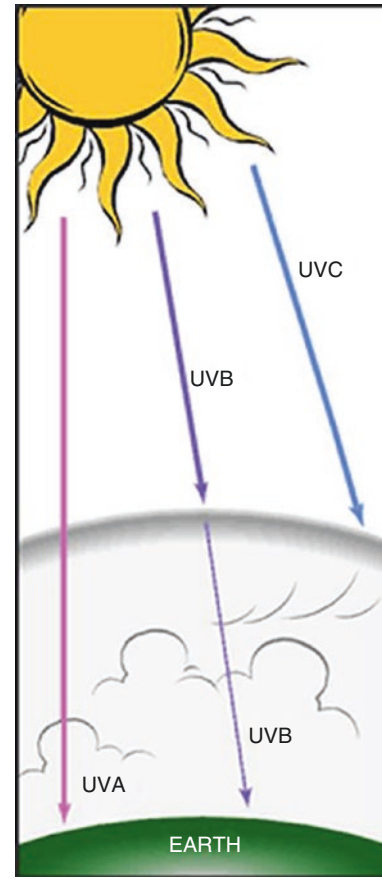


Fig. 14.8 High-energy UV-C radiation. Office of Air and Radiation (6205J); June 2010; EPA 430-F-10-025, The stratospheric ozone layer screens out much of the sun's harmful UV rays, http://www.epa.gov/sunwise/doc/uvradiation.html

Our eyes are constantly exposed to UV-induced photo-oxidation, generating free radical oxygen species which cause both acute oxidative injury and cumulative effects over time. Enhanced protein binding, biotransformation reactions, and a plethora of free radical scavengers such as glu-

tathione and superoxide dismutase are present in the eye to help compensate for these toxins.

Studies exploring corneal transmission of UV radiation as a function of position across the corneal surface show decreased transmission in the peripheral regions [172]. It is suspected that the effects of UV radiation are more pronounced in the posterior cornea (corneal endothelium), a single layer of cells that have little ability to proliferate in vivo. With maximum UV transmission in the central regions of the cornea, we would expect greater damage on cells through free radical oxidative injury. These mechanisms, in part, explain age-dependent injury patterns common to the central cornea.

Battlefield *laser* (light amplification by stimulated emission of radiation) injuries are increasing in frequency and deserve note. The use of powerful lasers for target identification and fire control is common on today's battlefield. Important factors in determining severity of injury are (1) location of retinal injury, (2) power output of the laser, and (3) duration of exposure [173]. A moderate retinal lesion outside the macula may be sub-clinical, whereas a small lesion within the fovea may be visually devastating.

Biological injury is determined by the amount of energy per unit area over a given exposure time (watts-sec/cm^3). Lasers are often classified by power output. Most 'laser pointers' in the United States are class 2 or 3a lasers (less than 5 mW output) [174, 175]. By contrast, class 3b (<500 mW) and class 4 (>500 mW) are typically used in occupational and military settings and capable of causing extensive retinal injury [176]. The retina is far more susceptible to laser injuries because the eye focuses light onto the retina with up to 10,000 times the irradiance present at the cornea [177]. Patients may present with cornea redness, pain, and irritation but any anterior structure injury is relatively minor and usually secondary to external rubbing or irritation. Spectral optical coherence tomography often demonstrates focal areas of retinal injury with inner and outer retinal segment disruption [178]. Patients may have "blind spots" in their visual field, but the presence and degree of impairment is often related to retinal lesion location in proximity to the fovea (central vision). Minor injuries usually recover over a period of weeks, but some patients develop chronic complications including progressive chorioretinal scarring, macular cyst/hole formation, epiretinal membrane formation, and CNV [179–181].

Definitive treatment of laser-induced retinal injuries is limited. There is support for systemic corticosteroids, antioxidants, and other anti-inflammatory agents. Intra-vitreal anti-vascular endothelial growth factors and steroid agents have been used to successfully treat radiation-induced macular edema and neovascular events secondary to radiation retinopathy in cancer patients, but visual outcomes in these patients remain variable [182, 183]. Future research will help evaluate the use of retinal-specific biomarkers for early detection of subtle or subclinical injuries.

Systemic Ocular Toxins

Beyond external exposures, systemic xenobiotics can impact vision via toxicity at many different sites within the eye. The proper function of each refractive segment of the eye is maintained by complex biochemical systems. These systems are vital both in the maintenance of tissue protein structure and metabolism, as well as detoxifying free radicals and other chemical stressors.

Many xenobiotics influence photo toxicity through the formation of stable ring structures which lead to free radical formation. Oxidation of thiol groups on lens proteins leads to cross-linking of polypeptide disulfide bonds and the accumulation of high molecular weight aggregates with enhanced light-scattering effects [184, 185]. Common examples include glucocorticoids which induce covalent modification of lens crystallins, followed by formation of water-insoluble aggregates or cataracts [186].

The retina is highly specialized and thus susceptible to xenobiotic mediated toxicity on many levels. Choriocapillaries with high flow rates allow diffusion of xenobiotics through loose junctions into the outer retina. Many xenobiotics are concentrated by melanin binding in the cho-

roid and RPE [187]. Oxidative toxicity is often synergistic between specific xenobiotics and UV-induced injury. High cellular metabolism may enhance retinal toxicity through accelerated production of secondary toxic metabolites [188].

Retinal neurotoxicity from lead poisoning has been well studied. Exposures in the United States have declined dramatically since the removal of lead from gasoline, but this is not the case for individuals working with jet fuel or military members overseas. Ocular pathology includes optic neuritis manifesting as decreased visual acuity, diplopia, ophthalmoplegia, amblyopia, and scotomas with progression to blindness [189]. Optic nerve toxicity from lead mimics CNS toxicity reflected in global neurocognitive deficits [190]. Early scotomas may only be apparent under scotopic conditions or with early ERG screening [191]. Animal studies suggest interference with cellular calcium signaling may cause mitochondrial homeostasis dysfunction leading to apoptosis [192]. A similar mechanism is proposed with other heavy metals including arsenic, thallium, and cobalt [193, 194].

Methanol, a widely used solvent and fuel source, causes irreversible retinal and optic nerve dysfunction secondary to its metabolism to formic acid. Visual impairment usually begins 12–24 hours post ingestion [195]. Although the exact mechanism is not known, it is suspected that both local and systemic metabolism to formic acid (formate) disrupts mitochondrial oxidative energy production. This proposed mechanism for optic neuropathy is echoed in other xenobiotics including chloramphenicol, ethambutol, carbon monoxide and cyanide [196, 200]. Malnourished individuals with folate deficiencies and decreased tetrahydrofolate activity are particularly sensitive to these effects. There is no definitive treatment for methanol-induced optic neuritis, but some studies report good outcomes with intravenous methylprednisolone [197].

Toxicity from systemic exposure to organic solvents, usually inhaled or dermal, is well recognized but the mechanism is not well understood. Dose-dependent dyschromatopsia and scotomas are well described in factory workers suffering from chronic exposures [198, 199].

Summary

Ocular chemical exposures should always be treated as true vision-threatening emergencies. Rapid and thorough decontamination is the mainstay of treatment and may be nothing short of vision saving. Healthcare providers need to be familiar with treatments of both occupational and insurgent-related or battlefield chemical exposures. Although ocular blast and open globe mechanisms dominate battlefield eye injuries, this can be partially mitigated by more consistent use of eye protection.

The presence of multiple eye or dermal complaints in a given proximity should alert providers to the possibility of CW agents. Education of current potential CW threats and proper use of PPE is requisite to all soldiers and LE personnel. Diluted hypochlorite solution (0.5% bleach) *may* be effective in decontamination of *intact* skin exposed to mustard vesicants and V-nerve agents but should not be used as ocular irrigation solutions. Ready-to-use commercial chemical decontaminants are highly effective against most CW agents, and include both dry and liquid formulations. When in doubt, copious soap and water is usually effective.

Beyond decontamination, early recognition of eye injuries is essential to help minimize long-term complications including delayed keratopathy, recurrent corneal ulceration, and secondary glaucoma. With regard to irrigation, tap water is at least as effective if not superior to saline rinse. Specific irrigation solutions such as DRS (diphoterine rinsing solution) and borate-buffered Cederroth Eye Wash have shown superior efficacy to standard saline in the setting of alkali and mustard eye exposures in animals. If these agents are not readily available, any neutral solutions prehospital (e.g., milk and water) is preferred to avoid any delays in immediate decontamination. The effects of RCAs, under proper use, are temporary and largely self-limited.

Poor visual outcomes after acute corneal injuries are associated with ongoing inflammation beyond the early healing phase (10–14 days). Close follow-up (sometimes daily) is required to optimize outcomes. Current studies support

the use of anti-inflammatory agents and immune modulators to aid in early healing, thereby minimizing sub-acute and chronic disease states that result in long-term visual disability. Ongoing early monitoring of IOP is essential to avoid secondary injury. Advances in surgical techniques including limbal stem cell and corneal transplantation have improved success rates in recent years. Discovery of injury-specific biochemical markers have expanded our understanding of these mechanisms.

Systemic exposure to xenobiotics frequently present with isolated neurologic or visual complaints. Healthcare providers should be aware of potential early symptoms including rapid changes in scotopic vision and dyschromatopsias. VEP and ERGs are valuable screening tools for suspected xenobiotic-induced retinal or macular injury in early or subclinical cases. When in doubt, early referral for comprehensive visual testing is recommended. Depending on exposure history, providers may refer patients to toxicology for further evaluation.

References

1. Weapons of war "poison gas" 2009. https://www.first-worldwar.com/weaponry/gas.htm.
2. OPCW: Report on the implementation of the convention on the prohibition of the development, production, stockpiling and use of chemical weapons and on their destruction. http://www.opcw.nl: OPCW; 2013.
3. Puangsricharern V, Tseng SC. Cytologic evidence of corneal diseases with limbal stem cell deficiency. Ophthalmology. 1995;102(10):1476–85.
4. Laibson PR, Oconor J. Explosive tear gas injuries of the eye. Trans Am Acad Ophthalmol Otolaryngol. 1970;74(4):811–9.
5. Adler. In: Adler, editor. Adler's physiology of the eye. 9th ed. St. Louis: Mosby-Year Book; 1992.
6. Sears. In: Sears, editor. Pharmacology of the eye: Springer-Verlag; 1984.
7. Eves P, Smith-Thomas L, Hedley S, Wagner M, Balafa C, Mac NS. A comparative study of the effect of pigment on drug toxicity in human choroidal melanocytes and retinal pigment epithelial cells. Pigment Cell Res. 1999;12(1):22–35.
8. Potts. Toxic responses of the eye Casarett and Doull's Toxicology: the basic science of poisons. 5th ed. New York: McGraw-Hill; 1996.
9. Goldsmith TH. Optimization, constraint, and history in the evolution of eyes. Q Rev Biol. 1990;65(3):281–322.
10. King G, Hirst L, Holmes R. Human corneal and lens aldehyde dehydrogenases. Localization and function(s) of ocular ALDH1 and ALDH3 isozymes. Adv Exp Med Biol. 1999;463:189–98.
11. Mader TH, Carroll RD, Slade CS, George RK, Ritchey JP, Neville SP. Ocular war injuries of the Iraqi Insurgency,January-September 2004. Ophthalmology. 2006;113(1):97–104.
12. CDC/DHHS. Explosions and blast injuries, a primer for clinicians; 2008.
13. Arya SK, Malhotra S, Dhir SP, Sood S. Ocular fireworks injuries. Clinical features and visual outcome. Indian J Ophthalmol. 2001;49(3):189–90.
14. Sacu S, Segur-Eltz N, Stenng K, Zehetmayer M. Ocular firework injuries at New Year's eve. Ophthalmologica. 2002;216(1):55–9.
15. Jr HW. Mustard gas injuries to the eyes. Arch Ophthalmol. 1942;(27):582–601.
16. Leopold IH, Lieberman TW. Chemical injuries of the cornea. Fed Proc. 1971;30(1):92–5.
17. Hoffmann DH. Eye burns caused by tear gas. Br J Ophthalmol. 1967;51(4):265–8.
18. Wang X, Zhang Y, Ni L, You C, Ye C, Jiang R, et al. A review of treatment strategies for hydrofluoric acid burns: current status and future prospects. Burns. 2014;40(8):1447–57.
19. Trevino MA, Herrmann GH, Sprout WL. Treatment of severe hydrofluoric acid exposures. J Occup Med. 1983;25(12):861–3.
20. Dohlman CH, Cade F, Pfister R. Chemical burns to the eye: paradigm shifts in treatment. Cornea. 2011;30(6):613–4.
21. Clare G, Suleman H, Bunce C, Dua H. Amniotic membrane transplantation for acute ocular burns. Cochrane Database Syst Rev. 2012;9:Cd009379.
22. Fish R, Davidson RS. Management of ocular thermal and chemical injuries, including amniotic membrane therapy. Curr Opin Ophthalmol. 2010;21(4):317–21.
23. Cade F, Paschalis EI, Regatieri CV, Vavvas DG, Dana R, Dohlman CH. Alkali burn to the eye: protection using TNF-alpha inhibition. Cornea. 2014;33(4):382–9.
24. Gupta N, Kalaivani M, Tandon R. Comparison of prognostic value of Roper Hall and Dua classification systems in acute ocular burns. Br J Ophthalmol. 2011;95(2):194–8.
25. Dua HS, King AJ, Joseph A. A new classification of ocular surface burns. Br J Ophthalmol. 2001;85(11):1379–83.
26. Suh MH, Kwon JW, Wee WR, Han YK, Kim JH, Lee JH. Protective effect of ascorbic Acid against corneal damage by ultraviolet B irradiation: a pilot study. Cornea. 2008;27(8):916–22.
27. Bunker DJ, George RJ, Kleinschmidt A, Kumar RJ, Maitz P. Alkali-related ocular burns: a case series and review. J Burn Care Res. 2014;35(3):261–8.
28. Geffen N, Topaz M, Kredy-Farhan L, Barequet IS, Farzam N, Assia EI, et al. Phacoemulsification-induced injury in corneal endothelial cells mediated

by apoptosis: in vitro model. J Cataract Refract Surg. 2008;34(12):2146–52.
29. Tewari-Singh N, Jain AK, Inturi S, Ammar DA, Agarwal C, Tyagi P, et al. Silibinin, dexamethasone, and doxycycline as potential therapeutic agents for treating vesicant-inflicted ocular injuries. Toxicol Appl Pharmacol. 2012;264(1):23–31.
30. Donshik PC, Berman MB, Dohlman CH, Gage J, Rose J. Effect of topical corticosteroids on ulceration in alkali-burned corneas. Arch Ophthalmol. 1978;96(11):2117–20.
31. Edward Trudo WR. Chapter 7 - Chemical Injuries of the Eye. Ophthalmic Care of the Combat Casualty Borden Institute, Office of the Surgeon General; 2003.
32. Spielmann H, Kalweit S, Liebsch M, Wirnsberger T, Gerner I, Bertram-Neis E, et al. Validation study of alternatives to the Draize eye irritation test in Germany: cytotoxicity testing and HET-CAM test with 136 industrial chemicals. Toxicol In Vitro. 1993;7(4):505–10.
33. Moldenhauer F. Using in vitro prediction models instead of the rabbit eye irritation test to classify and label new chemicals: a post hoc data analysis of the international EC/HO validation study. Altern Lab Anim. 2003;31(1):31–46.
34. Doucet O, Lanvin M, Thillou C, Linossier C, Pupat C, Merlin B, et al. Reconstituted human corneal epithelium: a new alternative to the Draize eye test for the assessment of the eye irritation potential of chemicals and cosmetic products. Toxicol In Vitro. 2006;20(4):499–512.
35. Ikarashi Y, Tsuchiya T, Nakamura A. Comparison of Three In Vitro Assays to Determine the Ocular Toxicity of Detergent, Oil, and Organic Solvents. Cutan Ocul Toxicol. 1993;12(1):15–24.
36. Sikkema J, de Bont JA, Poolman B. Mechanisms of membrane toxicity of hydrocarbons. Microbiol Rev. 1995;59(2):201–22.
37. Galvao J, Davis B, Tilley M, Normando E, Duchen MR, Cordeiro MF. Unexpected low-dose toxicity of the universal solvent DMSO. FASEB J. 2014;28(3):1317–30.
38. Lapalus P, Ettaiche M, Fredj-Reygrobellet D, Jambou D, Elena PP. Cytotoxicity studies in ophthalmology. Lens Eye Toxic Res. 1990;7(3–4):231–42.
39. DHHS/CDC/NIOSH. Guidance on Emergency Responder Personal Protective Equipment (PPE) for Response to CBRN Terrorism Incidents.: DHHS/CDC/NIOSH; 2008.
40. Taysse L, Daulon S, Delamanche S, Bellier B, Breton P. Skin decontamination of mustards and organophosphates: comparative efficiency of RSDL and Fuller's earth in domestic swine. Hum Exp Toxicol. 2007;26(2):135–41.
41. Trapp. The detoxification and natural degradation of chemical warfare agents: Stockholm International Peace Research Institute; 1985.
42. Braue EH Jr, Smith KH, Doxzon BF, Lumpkin HL, Clarkson ED. Efficacy studies of Reactive Skin Decontamination Lotion, M291 Skin Decontamination Kit, 0.5% bleach, 1% soapy water, and Skin Exposure Reduction Paste Against Chemical Warfare Agents, part 2: guinea pigs challenged with soman. Cutan Ocul Toxicol. 2011;30(1):29–37.
43. Kompa S, Redbrake C, Hilgers C, Wustemeyer H, Schrage N, Remky A. Effect of different irrigating solutions on aqueous humour pH changes, intraocular pressure and histological findings after induced alkali burns. Acta Ophthalmol Scand. 2005;83(4):467–70.
44. Chau JP, Lee DT, Lo SH. A systematic review of methods of eye irrigation for adults and children with ocular chemical burns. Worldviews Evid Based Nurs. 2012;9(3):129–38.
45. Hall AH, Blomet J, Mathieu L. Diphoterine for emergent eye/skin chemical splash decontamination: a review. Vet Hum Toxicol. 2002;44(4):228–31.
46. Schrage NF, Struck HG, Gerard M. Recommendations for acute treatment for chemical and thermal burns of eyes and lids. Ophthalmologe. 2011;108(10):916–20.
47. Goldich Y, Barkana Y, Zadok D, Avni I, Berenshtein E, Rosner M, et al. Use of amphoteric rinsing solution for treatment of ocular tissues exposed to nitrogen mustard. Acta Ophthalmol. 2013;91(1):e35–40.
48. Gerasimo PBJ, Mathieu L, Hall AH. Diphoterine decontamination of 14C-sulfur mustard contaminated human skin fragments in vitro. Toxicologist. 2000;(54):152.
49. Rihawi S, Frentz M, Reim M, Schrage NF. Rinsing with isotonic saline solution for eye burns should be avoided. Burns. 2008;34(7):1027–32.
50. Pless M, Friberg TR. Topical phenylephrine may result in worsening of visual loss when used to dilate pupils in patients with vaso-occlusive disease of the optic nerve. Semin Ophthalmol. 2003;18(4):218–21.
51. He M, Huang W, Friedman DS, Wu C, Zheng Y, Foster PJ. Slit lamp-simulated oblique flashlight test in the detection of narrow angles in Chinese eyes: the Liwan eye study. Invest Ophthalmol Vis Sci. 2007;48(12):5459–63.
52. Levinson. In: Levinson, editor. Clinical methods: the history, physical, and laboratory examinations. 3rd ed: Reed Publishing; 1990.
53. Blice JP. Ocular injuries, triage, and management in maxillofacial trauma. Atlas Oral Maxillofac Surg Clin North Am. 2013;21(1):97–103.
54. Norcia AM, Tyler CW, Allen D. Electrophysiological assessment of contrast sensitivity in human infants. Am J Optom Physiol Optic. 1986;63(1):12–5.
55. Regan D. Electrical responses evoked from the human brain. Sci Am. 1979;241(6):134–46.
56. Marmor MF. An updated standard for clinical electroretinography. Arch Ophthalmol. 1995;113(11):1375–6.
57. EPA. EPA: Health effect test guidelines. Neurophysiology: Sensory Evoked Potentials.: EPA; 1998.
58. Kollner. Die Störungen des Farbensinners. ihre klinische Bedeutung und ihre Diagnose. Karger; 1912.

59. Schwartz. Visual perception: a clinical orientation; 2004.
60. Department UW. U.S. War Department, General Orders No. 100, Adjutant General's Office, Washington, April 24. The War of the Rebellion: A Compilation of the Official Records of the Union and Confederate Armies. Washington DC: U.S. Government Printing Office; 1863. p. 1880–901.
61. CDC/NIOSH. Sulfur Mustard, CAS #: 505–60-2; RTECS #: WQ0900000; UN #: 2810. CDC/NIOSH; 2011.
62. Army US. Potential Military Chemical/Biological Agents and Compounds. US Army Field Manual 3–9, US Navy Publication P-467, US Air Force Manual 355–71990. p. 20,32.
63. Vidan A, Luria S, Eisenkraft A, Hourvitz A. Ocular injuries following sulfur mustard exposure: clinical characteristics and treatment. Isr Med Assoc J. 2002;4(7):577–8.
64. Graham JSSB. Historical perspective on effects and treatment of sulfur mustard injuries. Chem Biol Interact. 2013;206(3):512–22.
65. Grant W. In: Grant W, editor. Toxicology of the eye : effects on the eyes and visual system from chemicals, drugs, metals and minerals, plants, toxins and venoms. Springfield: Thomas; 1986.
66. Shulman LN. The biology of alkylating-agent cellular injury. Hematol Oncol Clin North Am. 1993;7(2):325–35.
67. Papirmeister B, Feister AJ, Robinson I, Ford RD (1991). Medical Defense Against Mustard Gas: Toxic Mechanisms and Pharmacological Implications. Boca Raton: CRC Press; 1991.
68. Javadi MA, Yazdani S, Kanavi MR, Mohammadpour M, Baradaran-Rafiee A, Jafarinasab MR, et al. Long-term outcomes of penetrating keratoplasty in chronic and delayed mustard gas keratitis. Cornea. 2007;26(9):1074–8.
69. McNutt P, Tuznik K, Nelson M, Adkins A, Lyman M, Glotfelty E, et al. Structural, morphological, and functional correlates of corneal endothelial toxicity following corneal exposure to sulfur mustard vapor. Invest Ophthalmol Vis Sci. 2013;54(10):6735–44.
70. Kadar T, Dachir S, Cohen L, Sahar R, Fishbine E, Cohen M, et al. Ocular injuries following sulfur mustard exposure--pathological mechanism and potential therapy. Toxicology. 2009;263(1):59–69.
71. Rama P, Matuska S, Paganoni G, Spinelli A, De Luca M, Pellegrini G. Limbal stem-cell therapy and long-term corneal regeneration. N Engl J Med. 2010;363(2):147–55.
72. Frank MH, Frank NY. Restoring the cornea from limbal stem cells. Regen Med. 2015;10(1):1–4.
73. Gilman A. The initial clinical trial of nitrogen mustard. Am J Surg. 1963;105:574–8.
74. Atkins KB, Lodhi IJ, Hurley LL, Hinshaw DB. N-acetylcysteine and endothelial cell injury by sulfur mustard. J Appl Toxicol. 2000;20(Suppl 1):S125–8.
75. Shohrati M, Karimzadeh I, Saburi A, Khalili H, Ghanei M. The role of N-acetylcysteine in the management of acute and chronic pulmonary complications of sulfur mustard: a literature review. Inhal Toxicol. 2014;26(9):507–23.
76. Jugg B, Fairhall S, Smith A, Rutter S, Mann T, Perrott R, et al. N-acetyl-L-cysteine protects against inhaled sulfur mustard poisoning in the large swine. Clin Toxicol (Phila). 2013;51(4):216–24.
77. Devereaux A, Amundson DE, Parrish JS, Lazarus AA. Vesicants and nerve agents in chemical warfare. Decontamination and treatment strategies for a changed world. Postgrad Med. 2002;112(4):90–6; quiz 4.
78. Vijayaraghavan R, Kumar P, Joshi U, Raza SK, Lakshmana Rao PV, Malhotra RC, et al. Prophylactic efficacy of amifostine and its analogues against sulphur mustard toxicity. Toxicology. 2001;163(2–3):83–91.
79. Sidell FRUJ, Smith WJ. Part I: Medical aspects of chemical and biological warfare. In: Zajtchuk B, editor. Textbook of military medicine. Falls Church: Office of the Surgeon General, Dept of the Army; 1997. p. 197–228.
80. Gupta RC. Handbook of toxicology of chemical warfare agents: Academic Press; 2009.
81. Gates MWJ, Zapp JA. In: Committee NDR, editor. Arsenicals: chemical warfare agents and related chemical problems. Washington, D.C; 1946.
82. IOM. Institute of Medicine Committee on the Survey of the Health. Effects of mustard gas and lewisite. In: Pechura CM, Rall DP, editors. Veterans at Risk: the health effects of mustard gas and lewisite. Washington, DC: National Academies Press (US) Copyright 1993 by the National Academy of Sciences. All rights reserved; 1993.
83. Hughes WF Jr. Clinical uses of 2,3-dimercaptopropanol (BAL); the treatment of lewisite burns of the eye with BAL. J Clin Invest. 1946;25(4):541–8.
84. Aaseth J, Skaug MA, Cao Y, Andersen O. Chelation in metal intoxication-Principles and paradigms. J Trace Elem Med Biol. 2015;31:260–6.
85. Andersen O. Principles and recent developments in chelation treatment of metal intoxication. Chem Rev. 1999;99(9):2683–710.
86. Evans RB. Chlorine: state of the art. Lung. 2005;183(3):151–67.
87. Massa CB, Scott P, Abramova E, Gardner C, Laskin DL, Gow AJ. Acute chlorine gas exposure produces transient inflammation and a progressive alteration in surfactant composition with accompanying mechanical dysfunction. Toxicol Appl Pharmacol. 2014;278(1):53–64.
88. Bismuth C, Borron SW, Baud FJ, Barriot P. Chemical weapons: documented use and compounds on the horizon. Toxicol Lett. 2004;149(1–3):11–8.
89. CDC/ATSDR. Medical Management Guidelines for Chlorine, CAS# 7782-50-5, UN# 1017. In: DHHS/CDC/ATSDR, editor.: CDC/ATSDR; 2014.
90. Wang J, Winskog C, Edston E, Walther SM. Inhaled and intravenous corticosteroids both attenuate chlorine gas-induced lung injury in pigs. Acta Anaesthesiol Scand. 2005;49(2):183–90.

91. Gunnarsson M, Walther SM, Seidal T, Lennquist S. Effects of inhalation of corticosteroids immediately after experimental chlorine gas lung injury. J Trauma. 2000;48(1):101–7.
92. Vinsel PJ. Treatment of acute chlorine gas inhalation with nebulized sodium bicarbonate. J Emerg Med. 1990;8(3):327–9.
93. CalEPA. Air Toxics Hot Spots Program Risk Assessment Guidelines: Part III. In: Assessment OoEHH, editor. Technical Support Document for the Determination of Noncancerous Chronic Reference Exposure Levels. SRP Draft. Berkeley: California Environmental Protection Agency; 1999.
94. Van Sickle D, Wenck MA, Belflower A, Drociuk D, Ferdinands J, Holguin F, et al. Acute health effects after exposure to chlorine gas released after a train derailment. Am J Emerg Med. 2009;27(1):1–7.
95. Jones R, Wills B, Kang C. Chlorine gas: an evolving hazardous material threat and unconventional weapon. West J Emerg Med. 2010;11(2):151–6.
96. Luo S, Trubel H, Wang C, Pauluhn J. Phosgene- and chlorine-induced acute lung injury in rats: comparison of cardiopulmonary function and biomarkers in exhaled breath. Toxicology. 2014;326:109–18.
97. CDC/DHHS. Phosgene: Emergency Preparedness and Response.: CDC/DHHS; 2013.
98. Jugg B, Jenner J, Rice P. The effect of perfluoroisobutene and phosgene on rat lavage fluid surfactant phospholipids. Hum Exp Toxicol. 1999;18(11):659–68.
99. Li W, Liu F, Wang C, Truebel H, Pauluhn J. Novel insights into phosgene-induced acute lung injury in rats: role of dysregulated cardiopulmonary reflexes and nitric oxide in lung edema pathogenesis. Toxicol Sci. 2013;131(2):612–28.
100. Pauluhn J, Hai CX. Attempts to counteract phosgene-induced acute lung injury by instant high-dose aerosol exposure to hexamethylenetetramine, cysteine or glutathione. Inhal Toxicol. 2011;23(1):58–64.
101. Li W, Rosenbruch M, Pauluhn J. Effect of PEEP on phosgene-induced lung edema: pilot study on dogs using protective ventilation strategies. Exp Toxicol Pathol. 2015;67(2):109–16.
102. Vaish AK, Consul S, Agrawal A, Chaudhary SC, Gutch M, Jain N, et al. Accidental phosgene gas exposure: A review with background study of 10 cases. J Emerg Trauma Shock. 2013;6(4):271–5.
103. Thomas C. In: Grant W, editor. Toxicology of the eye. 2nd ed; 1974.
104. Hygienists ACoGI, editor Documentation of the TLV's and BEI's with Other World Wide Occupational Exposure Values. American Conference of Governmental Industrial Hygienists 2006. Cincinnati, OH.
105. CDC/ATSDR. Medical Management Guidelines for Phosgene (COCl2), CAS# 75–44-5, UN# 1076. CDC/ATSDR; 2014.
106. Diller WF. Early diagnosis of phosgene overexposure. Toxicol Ind Health. 1985;1(2):73–80.
107. Kundu P, Hwang KC. Rational design of fluorescent phosgene sensors. Anal Chem. 2012;84(10):4594–7.
108. Hulse EJ, Davies JO, Simpson AJ, Sciuto AM, Eddleston M. Respiratory complications of organophosphorus nerve agent and insecticide poisoning. Implications for respiratory and critical care. Am J Respir Crit Care Med. 2014;190(12):1342–54.
109. Quistad GB, Sparks SE, Casida JE. Fatty acid amide hydrolase inhibition by neurotoxic organophosphorus pesticides. Toxicol Appl Pharmacol. 2001;173(1):48–55.
110. Okumura T, Hisaoka T, Naito T, Isonuma H, Okumura S, Miura K, et al. Acute and chronic effects of sarin exposure from the Tokyo subway incident. Environ Toxicol Pharmacol. 2005;19(3):447–50.
111. Chandar NB, Ganguly B. A first principles investigation of aging processes in soman conjugated AChE. Chem Biol Interact. 2013;204(3):185–90.
112. Foltin G, Tunik M, Curran J, Marshall L, Bove J, van Amerongen R, et al. Pediatric nerve agent poisoning: medical and operational considerations for emergency medical services in a large American city. Pediatr Emerg Care. 2006;22(4):239–44.
113. AAP. Chemical-biological terrorism and its impact on children: a subject review. American Academy of Pediatrics. Committee on Environmental Health and Committee on Infectious Diseases. Pediatrics. 2000;105(3 Pt 1):662–70.
114. Chung S, Shannon M. Hospital planning for acts of terrorism and other public health emergencies involving children. Arch Dis Child. 2005;90(12):1300–7.
115. Eddleston M, Roberts D, Buckley N. Management of severe organophosphorus pesticide poisoning. Crit Care. 2002;6(3):259.
116. Sternbach LHaSK. Antispasmodics: (1)Bicyclic basic alcohols, (2)Esters of basic bicyclic alcohols. J Am Chem Soc. 1952;74.
117. Corps USAC. Joint CB Technical Data Source Book: Volume II Riot Control and Incapacitating Agents, Part Three: Agent BZ. Fort Douglas, Utah; 1972.
118. Rosenblatt D, Dacre J, Shiotsuka R, Rowlett C. Problem definition studies on the potential environmental pollutants VIII. Chemistry and toxicology of BZ (3-Quinuclidinyl Benzinlate), TR 7710. In: Army U, editor. . Fort Detrick: US Army Medical Bioengineering Research and Development Laboratory; 1977.
119. US Army BI. Chapter 5: incapacitating agents. In: Medical management of chemical casualties handbook: US Army, Borden Institute.
120. Ballantyne B, Swanston DW. The comparative acute mammalian toxicity of 1-chloroacetophenone (CN) and 2-chlorobenzylidene malononitrile (CS). Arch Toxicol. 1978;40(2):75–95.
121. Chapman AJ, White C. Death resulting from lacrimatory agents. J Forensic Sci. 1978;23(3):527–30.
122. Rothberg S. Skin sensitization potential of the riot control agents BBC, DM, CN and CS in guinea pigs. Mil Med. 1970;135(7):552–6.
123. Ballantyne B, Callaway S. Inhalation toxicology and pathology of animals exposed to

o-chlorobenzylidene malononitrile (CS). Med Sci Law. 1972;12(1):43–65.
124. Gaskins JR, Hehir RM, McCaulley DF, Ligon EW Jr. Lacrimating agents (CS and CN) in rats and rabbits. Acute effects on mouth, eyes, and skin. Arch Environ Health. 1972;24(6):449–54.
125. Rengstorff RH, Mershon MM. CS in trioctyl phosphate: effects on human eyes. Mil Med. 1971;136(2):152–3.
126. Ballantyne B, Gazzard MF, Swanston DW, Williams P. The ophthalmic toxicology of o-chlorobenzylidene malononitrile (CS). Arch Toxicol. 1974;32(3):149–68.
127. Beswick FW, Holland P, Kemp KH. Acute effects of exposure to orthochlorobenzylidene malononitrile (CS) and the development of tolerance. Br J Ind Med. 1972;29(3):298–306.
128. Kluchinsky TA Jr, Sheely MV, Savage PB, Smith PA. Formation of 2-chlorobenzylidenemalononi trile (CS riot control agent) thermal degradation products at elevated temperatures. J Chromatogr A. 2002;952(1–2):205–13.
129. Kluchinsky TA Jr, Savage PB, Fitz R, Smith PA. Liberation of hydrogen cyanide and hydrogen chloride during high-temperature dispersion of CS riot control agent. AIHA J (Fairfax, Va). 2002;63(4):493–6.
130. EPA. The Environmental Protection Agency's (EPA) List of Registered Bear Deterrents containing capsaicin (regulated under FIFRA). EPA; 1996.
131. Peter KV. In: Peter KV, editor. Handbook of herbs and spices: Woodhead Publishing; 2012.
132. Tainter D, Grenis A, Norwat R. Spices and seasonings (A Food Technology Handbook). Second edition. Food Serv Technol. 2001;1:181. https://doi.org/10.1046/j.1471-5740.2001.d01-1.x.
133. Reilly CA, Crouch DJ, Yost GS. Quantitative analysis of capsaicinoids in fresh peppers, oleoresin capsicum and pepper spray products. J Forensic Sci. 2001;46(3):502–9.
134. Weiser T, Roufogalis B, Chrubasik S. Comparison of the effects of pelargonic acid vanillylamide and capsaicin on human vanilloid receptors. Phytother Res. 2013;27(7):1048–53.
135. Kozukue N, Han JS, Kozukue E, Lee SJ, Kim JA, Lee KR, et al. Analysis of eight capsaicinoids in peppers and pepper-containing foods by high-performance liquid chromatography and liquid chromatography-mass spectrometry. J Agric Food Chem. 2005;53(23):9172–81.
136. Steffee CH, Lantz PE, Flannagan LM, Thompson RL, Jason DR. Oleoresin capsicum (pepper) spray and "in-custody deaths". Am J Forensic Med Pathol. 1995;16(3):185–92.
137. Gosselin RE, Hodge HC, Smith RP, Gleason MN. Clinical toxicology of commercial products. 4th ed. Baltimore: Williams and Wilkins; 1976.
138. Monsereenusorn Y. Subchronic toxicity studies of capsaicin and capsicum in rats. Res Commun Chem Pathol Pharmacol. 1983;41(1):95–110.
139. toxicology Ijo. Final report on the safety assessment of *capsicum annuum* extract, *capsicum annuum* fruit extract, *capsicum annuum* resin, *capsicum annuum* fruit powder, *capsicum frutescens* fruit, *capsicum frutescens* fruit extract, *capsicum frutescens* resin, and capsaicin. Int J Toxicol. 2007;26(Suppl 1):3–106.
140. Hazari MS, Rowan WH, Winsett DW, Ledbetter AD, Haykal-Coates N, Watkinson WP, et al. Potentiation of pulmonary reflex response to capsaicin 24h following whole-body acrolein exposure is mediated by TRPV1. Respir Physiol Neurobiol. 2008;160(2):160–71.
141. E T-H. Cough reduction using capsaicin. Respir Med. 2015;109(1):27–37.
142. Tuorinsky SD. Medical aspects of chemical warfare.: Office of the Surgeon General, Department of the Army. Washington, D.C: Borden Institute (U.S.) Government Printing Office; 2008.
143. Voegeli S, Baenninger PB. Severe chemical burn to the eye after pepper spray attack. Klin Monbl Augenheilkd. 2014;231(4):327–8.
144. Fujita S, Shimizu T, Izumi K, Fukuda T, Sameshima M, Ohba N. Capsaicin-induced neuroparalytic keratitis-like corneal changes in the mouse. Exp Eye Res. 1984;38(2):165–75.
145. Gerber S, Frueh BE, Tappeiner C. Conjunctival proliferation after a mild pepper spray injury in a young child. Cornea. 2011;30(9):1042–4.
146. Olajos EJ, Salem H. Riot control agents: pharmacology, toxicology, biochemistry and chemistry. J Appl Toxicol. 2001;21(5):355–91.
147. Ballantyne B, Beswick FW, Thomas DP. The presentation and management of individuals contaminated with solutions of dibenzoxazepine (CR). Med Sci Law. 1973;13(4):265–8.
148. Ballantyne B, Swanston DW. The irritant effects of dilute solutions of dibenzoxazepine (CR) on the eye and tongue. Acta Pharmacol Toxicol. 1974;35(5):412–23.
149. Ashton I, Cotes JE, Holland P, Johnson GR, Legg SJ, Saunders MJ, et al. Acute effect of dibenz b.f.--1:4 oxazepine aerosol upon the lung function of healthy young men [proceedings]. J Physiol. 1978; 275:85.
150. Colgrave HF, Brown RF, Cox RA. Ultrastructure of rat lungs following exposure to aerosols of dibenzoxazepine (CR). Br J Exp Pathol. 1979;60(2):130–41.
151. Ballantyne B, Gazzard MF, Swanston DW, Williams P. The comparative ophthalmic toxicology of 1-chloroacetophenone (CN) and dibenz(b.f)-1:4-oxazepine(CR). Arch Toxicol. 1975;34(3):183–201.
152. Stenhouse J, editor. On the economical applications of Charcoal to sanitary purposes, notices of the proceedings at the meetings of the Members of the Royal Institution of Great Britain; 1855.
153. EPA. RED Fact Sheet: Chloropicrin. US EPA: EPA; 2008.
154. CDC/NIOSH. CHLOROPICRIN (PS) : Lung Damaging Agent; CAS #: 76–06-2; RTECS #: PB6300000; UN #: 1580. CDC/NIOSH; 2014.

155. Sparks SE, Quistad GB, Casida JE. Chloropicrin: reactions with biological thiols and metabolism in mice. Chem Res Toxicol. 1997;10(9):1001–7.
156. Pesonen M, Hakkinen M, Rilla K, Juvonen R, Kuitunen T, Pasanen M, et al. Chloropicrin-induced toxic responses in human lung epithelial cells. Toxicol Lett. 2014;226(2):236–44.
157. Pesonen M, Pasanen M, Loikkanen J, Naukkarinen A, Hemmila M, Seulanto H, et al. Chloropicrin induces endoplasmic reticulum stress in human retinal pigment epithelial cells. Toxicol Lett. 2012;211(3):239–45.
158. OEHHA C. Acute RELs and toxicity summaries using the previous version of the Hot Spots Risk Assessment Guidelines: CA OEHHA; 1999.
159. DHHS/NIOSH. NIOSH Pocket Guide to Chemical Hazards, National Institute for Occupational Safety and Health (NIOSH) Education and Information Division: DHHS/NIOSH; 2015.
160. CDC. Brief report: exposure to tear gas from a theft-deterrent device on a safe--Wisconsin, December 2003. MMWR Morb Mortal Wkly Rep. 2004;53(8):176–7.
161. Zasshi ONeaNNI. Case Report: Chloropicrin Eye Exposure. Japan Assoc Rural Med, Toxnet. 1980;29(3).
162. Prentiss A. Chemicals in war; a treatise on chemical warfare. New York/London: McGraw-Hill Book Company; 1937.
163. Hersh SM. Chemical and biological warfare: America's hidden arsenal: Doubleday; 1969.
164. McNamara BPOE, Weimer JT, Ballard TA. Toxicology of riot control chemicals - CS, CN and DM, EDGEWOOD ARSENAL ABERDEEN PROVING GROUND; Apr 1965-Jul 1968. 1968.
165. Owens M. Toxicology of DM, DEPARTMENT OF THE ARMY EDGEWOOD ARSENAL, Oct 1967. Maryland: Edgewood Arsenal; 1967.
166. Ltd O. http://www.skunk-skunk.com/121755/The-Product Aviezer 121/1 99860 Israel.
167. Ltd O. MSDS Skunk; http://www.skunk-skunk.com/image/users/121755/ftp/my_files/MSDS_Skunk.pdf?id=3225191.
168. Wennig R, Schneider S, Meys F. GC/MS based identification of skunk spray maliciously deployed as "biological weapon" to harm civilians. J Chromatogr B Analyt Technol Biomed Life Sci. 2010;878(17–18):1433–6.
169. CDC/NIOSH. NIOSH Guide to Chemical Hazards: N-butyl Mercaptan.: CDC/NIOSH; 2015.
170. Fierro BR, Agnew DW, Duncan AE, Lehner AF, Scott MA. Skunk musk causes methemoglobin and Heinz body formation in vitro. Vet Clin Pathol. 2013;42(3):291–300.
171. Zaks KL, Tan EO, Thrall MA. Heinz body anemia in a dog that had been sprayed with skunk musk. J Am Vet Med Assoc. 2005;226(9):1516–8.
172. Starr C. Biology: concepts and applications. Belmont: Thomson Brooks/Cole; 2006.
173. Doutch JJ, Quantock AJ, Joyce NC, Meek KM. Ultraviolet light transmission through the human corneal stroma is reduced in the periphery. Biophys J. 2012;102(6):1258–64.
174. Barkana Y, Belkin M. Laser eye injuries. Surv Ophthalmol. 2000;44(6):459–78.
175. Marshall J. The safety of laser pointers: myths and realities. Br J Ophthalmol. 1998;82(11):1335–8.
176. Lamotte J, Fife J, Lee A, Hemenger R. The power output of laser pointers: do they exceed federal standards? Optom Vis Sci. 2001;78(7):525–8.
177. Boosten K, Van Ginderdeuren R, Spileers W, Stalmans I, Wirix M, Van Calster J, et al. Laser-induced retinal injury following a recreational laser show: two case reports and a clinicopathological study. Bull Soc Belge Ophtalmol. 2011;317:11–6.
178. Albert DM. In: Albert DM, editor. Principles and practice of ophthalmology. 3rd ed: Saunders; 2008.
179. Giani A, Thanos A, Roh MI, Connolly E, Trichonas G, Kim I, et al. In vivo evaluation of laser-induced choroidal neovascularization using spectral-domain optical coherence tomography. Invest Ophthalmol Vis Sci. 2011;52(6):3880–7.
180. Owens SL, Bunce C, Brannon AJ, Wormald R, Bird AC. Prophylactic laser treatment appears to promote choroidal neovascularisation in high-risk ARM: results of an interim analysis. Eye (Lond). 2003;17(5):623–7.
181. Montezuma SR, Vavvas D, Miller JW. Review of the ocular angiogenesis animal models. Semin Ophthalmol. 2009;24(2):52–61.
182. Nakajima T, Hirata M, Shearer TR, Azuma M. Mechanism for laser-induced neovascularization in rat choroid: accumulation of integrin alpha chain-positive cells and their ligands. Mol Vis. 2014;20:864–71.
183. Reichstein D. Current treatments and preventive strategies for radiation retinopathy. Curr Opin Ophthalmol. 2015;26(3):157–66.
184. Aslam SA, Davies WI, Singh MS, Charbel Issa P, Barnard AR, Scott RA, et al. Cone photoreceptor neuroprotection conferred by CNTF in a novel in vivo model of battlefield retinal laser injury. Invest Ophthalmol Vis Sci. 2013;54(8):5456–65.
185. Lou MF. Redox regulation in the lens. Prog Retin Eye Res. 2003;22(5):657–82.
186. Raghavachari N, Qiao F, Lou MF. Does glutathione-S-transferase dethiolate lens protein-thiol mixed disulfides?-A comparative study with thioltransferase. Exp Eye Res. 1999;68(6):715–24.
187. Wang L, Zhao WC, Yin XL, Ge JY, Bu ZG, Ge HY, et al. Lens proteomics: analysis of rat crystallins when lenses are exposed to dexamethasone. Mol BioSyst. 2012;8(3):888–901.
188. Dayhaw-Barker P. Retinal pigment epithelium melanin and ocular toxicity. Int J Toxicol. 2002;21(6):451–4.

189. Metry KJ, Neale JR, Doll MA, Howarth AL, States JC, McGregor WG, et al. Effect of rapid human N-acetyltransferase 2 haplotype on DNA damage and mutagenesis induced by 2-amino-3-methylimidazo-[4,5-f]quinoline (IQ) and 2-amino-3,8-dimethylimidazo-[4,5-f]quinoxaline (MeIQx). Mutat Res. 2010;684(1–2):66–73.
190. Brown DV. Reaction of the rabbit retinal pigment ipithelium to systemic lead poisoning. Trans Am Ophthalmol Soc. 1974;72:404–47.
191. You Y, Gupta VK, Li JC, Klistorner A, Graham SL. Optic neuropathies: characteristic features and mechanisms of retinal ganglion cell loss. Rev Neurosci. 2013;24(3):301–21.
192. Ruther K, Foerster J, Berndt S, Schroeter J. Chloroquine/hydroxychloroquine: variability of retinotoxic cumulative doses. Ophthalmologe. 2007;104(10):875–9.
193. Yang P, Baciu P, Kerrigan BC, Etheridge M, Sung E, Toimil BA, et al. Retinal pigment epithelial cell death by the alternative complement cascade: role of membrane regulatory proteins, calcium, PKC, and oxidative stress. Invest Ophthalmol Vis Sci. 2014;55(5):3012–21.
194. Bahiga LM, Kotb NA, El-Dessoukey EA. Neurological syndromes produced by some toxic metals encountered industrially or environmentally. Z Ernahrungswiss. 1978;17(2):84–8.
195. Apel W, Stark D, Stark A, O'Hagan S, Ling J. Cobalt-chromium toxic retinopathy case study. Doc Ophthalmol. 2013;126(1):69–78.
196. Sanaei-Zadeh H, Zamani N, Shadnia S. Outcomes of visual disturbances after methanol poisoning. Clin Toxicol (Phila). 2011;49(2):102–7.
197. Carelli V, Ross-Cisneros FN, Sadun AA. Optic nerve degeneration and mitochondrial dysfunction: genetic and acquired optic neuropathies. Neurochem Int. 2002;40(6):573–84.
198. Shah S, Pandey V, Thakore N, Mehta I. Study of 63 cases of methyl alcohol poisoning (hooch tragedy in Ahmedabad). J Assoc Physicians India. 2012;60:34–6.
199. Mergler D, Blain L. Assessing color vision loss among solvent-exposed workers. Am J Ind Med. 1987;12(2):195–203.
200. Cavalleri A, Gobba F, Nicali E, Fiocchi V. Dose-related color vision impairment in toluene-exposed workers. Arch Environ Health. 2000;55(6):399–404.
201. Roper-Hall MJ. Thermal and chemical burns. Trans Ophthalmol Soc U K. 1965;85:631–53.
202. Ivarsson U, Nilsson H, Santesson J, editors. A FOA briefing book on chemical weapons: threat, effects, and protection. Umeå: National Defence Research Establishment; 1992.
203. Kayama M, Kurokawa MS, Ueno H, Suzuki N. Recent advances in corneal regeneration and possible application of embryonic stem cell-derived corneal epithelial cells. Clin Ophthalmol. 2007;1(4):373–82.

15 Winning the Hearts and Minds: Ophthalmology

Robert W. Enzenauer, Francis G. La Piana, W. Dale Anderson, and Warner D. "Rocky" Farr

As enumerated by Col Kimberly K. Armstrong in her 2007 US Army War College thesis, *Army Medical Department Support to Stability Operations*, during "the 1898 Spanish American War, army medical personnel were employed in widespread efforts to control contagious diseases in Cuba, Puerto Rico, and the Philippines. Direct care was provided to the civilian population and an extensive public health campaign was initiated to eliminate bubonic plague, vaccinate against smallpox, and institute measures for a safe water supply. Lieutenant General Arthur MacArthur, military governor of the Philippines, 'Felt that medical care was significant in winning over the urban population, depriving the guerillas of their support base and supplies necessary to continue the fight and securing victory" [1]. It is no coincidence that the most successful public health reforms in Cuba after cessation of hostilities were organized by physician turned brigadier general of volunteers Leonard Wood, appointed the Military Governor of Santiago in 1898 and of Cuba from 1899 to 1902. He relied on his medical experience to institute improvements in the medical and sanitary conditions of Cuba.

Recent doctrine describes counterinsurgency (COIN) as a comprehensive effort designed to simultaneously defeat and contain insurgency and address its root causes [2]. According to FM 3-24.2, the US Army thinking and doctrine on COIN tactics since the end of World War II have focused on the conduct of counterguerrilla operations in the later stages of insurgency. The Army has seen itself as defeating guerilla forces – usually communist forces – rather than defeating an entire insurgency. It was a success that could be achieved by using the force of the conventional army directly against guerilla forces. This doctrine of COIN began to take shape shortly after World War II in manuals such as ST 31-20-1, *Operations against Guerrilla Forces* (1950), FM 31-21, *Organization and Conduct of Guerilla Warfare* (1951), and later in FM 31-15, *Operations against Irregular Forces* (1961), FM 31-16, *Counterguerilla Operations* 1963). The Army refined its counterinsurgency doctrine during Vietnam in FM 31-22, *US Army Counterinsurgency Forces*, FM 31-16, *Counterguerilla Operations*, FM 31-22

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*US Counterinsurgency Forces (*all published in 1963), FM 31-20 *Special Forces Operational Techniques* (1965), and in FM 31-23, *Stability Operations in US Army Doctrine* (1972). After Vietnam, the Army split COIN doctrine off from conventional "high intensity" operations in FM 90-8, *Counterguerrilla Operations* (1986), FM 100-20/AFP 3-20, *Military Operations in Low Intensity Conflict* (1981, 1990), FM 7-98, *Operations in a Low-Intensity Conflict* 1992), FM 3-07 (FM 100-20) *Stability Operations and Support Operations* (2003) in which the "light forces" owned counterinsurgency, and FM 90-8, *Counterguerilla Operations* (1986), where the focus remained fixed on defeating the guerilla force. At its heart, a counterinsurgency is an armed struggle for the support of the population [3]. The Global War on Terror began on September 11, 2001. It is probably no coincidence that the Department of Defense published *Irregular Warfare (IW) Joint Operating Concept (JOC)* on 11 September 2007.

Successful COIN operations require competence and judgment at all levels. Indeed, young leaders often make decisions at a tactical level that have strategic consequences [4]. In the words of Australian Lt Gen Peter Leahy, "The era of the strategic corporal is here. The soldier of today must possess professional mastery of warfare, but match this with political and media sensitivity" [5]. Historical examples of successful guerillas include the *Maquis* in World War II occupied France, the *Viet Cong* in the Vietnam War, and the *Madhi* army in Iraq [3].

Although there is not clear evidence of "winning the hearts and minds" ophthalmologic activities in World War I and World War II, it has always been sound military policy to respond to any emergency patient presenting with injuries threatening "life, limb, or eyesight." During World War I, "In the British service," one capable ophthalmic surgeon in ordinary times was able to take care of the eye cases of about five general hospitals (1500–2000 beds), while in times of sustained combat probably two surgeons were needed. During American involvement in World War I, about 15 ophthalmic surgeons were assigned to the six eye centers created on the European continent for eye patients, aggregating about 500 beds from approximately 50,000 total hospital beds. However, in describing "Ophthalmology in the American Expeditionary Forces" Col (Dr) Allan Greenwood reports, "In the out-patient department some trachoma is noted, chiefly among collie laborers; other cases are found in inspecting tours."Greenwood summarized, "A resume of the clinical work of the ophthalmologists in the American Expeditionary Forces would not be complete without some reference to their work outside of their special field. Many of these officers did yeoman service as surgical assistants or as dressers in the wards, or even took charge of surgical cases both in the operating room and in the wards." A formal directive was sent from the headquarters of the medical and surgical consultants to the commanding officers of hospitals, directing "that the ophthalmic surgeons who are appointed to mobile and evacuation hospitals be assigned as regular assistants one of the permanent surgical teams, their duties to be those of the regular surgical assistants, except in cases of ocular wounds and injuries, when the chief of the team will in turn assist the ophthalmic surgeon in the performance of the necessary eye operation" [6].

During World War II, the experiences of ophthalmic surgeons were extensively catalogued by M. Elliott Randolph, MD. The activities of military eye surgeons were markedly different in the Zone of the Interior compared with the European Theater of Operations in contrast to the Southwest Pacific and Pacific Ocean areas. However, staffing of qualified and experienced eye surgeons was hampered by the fact that the TO&E, the Table of Organization and Equipment of a hospital in World War II was a carryover from World War I, when a single medical officer was responsible for both ophthalmology and otolaryngology, thus missing the evolution into two separate specialties between the world wars. US ophthalmologists were not assigned as far forward as the field hospitals since it was felt that ophthalmologists assigned to evacuation hospitals were not completely occupied with duties of their specialty, largely because of the lack of refraction equipment. No eye surgeons were assigned to station hospitals. Eye casualties could be evacuated to general hospitals that were

normally within consultation range. However, combat activity often resulted in significant delay in moving such patients. Interestingly, the British "solved" this problem through the creation of 6–8 mobile ophthalmologic units consisting of optical and surgical cans and an assigned ophthalmologist and other personnel and deployed as action demanded. Interestingly, some aspects of military bureaucracy would be readily recognized by currently serving medical officers. Based on a 1945 survey, some wastage of trained personnel was identified. Two board certified ophthalmologists were assigned to nonmedical units of the Fifth US Army in accordance with the policy of giving all recently arrived medical officers 6 months of field training before assigning them to hospitals. In addition, numerous ophthalmologists assigned to Army Air Force units were also doing little or no work in their specialty. In addition, the ophthalmic experience and training was lost in many instances, because of the fact that promotion beyond the rank of major was possible only by their assumption of duties not related to the specialty. Well-qualified ophthalmologists were thus lost to clinical ophthalmology because they naturally took the only available means of gaining additional rank and the associated increases in pay and allowances [7].

During the Korean War, there was generally only one eye surgeon-ophthalmologist "in theater," and he would routinely move from hospital to hospital depending upon where the casualties were piling up, depending upon where the eye casualties needed surgery. Based on an interview with a Korean War veteran ophthalmologist, it is not likely that a solo eye surgeon had the opportunity to practice much civic action type ophthalmology.

The Vietnam War was America's first armed conflict where "civic action" was formally planned and executed. As noted by Dr. Wilensky, the medical services have long been an integral part of armies at war. However, prior to this 1984 publication, the care delivered to civilians has not been extensively studied. Moreover, Wilensky's comprehensive study of the various programs providing medical care to Vietnamese civilians during the war was only published in 2004. According to Wilensky, the provision of civilian medical care by military personnel could be attributed to multiple possible motivations: (1) pure altruism, the simple desire to help those in need; (2) for strategic purpose, to win "the hearts and minds" of the people; and (3) most controversial, the acquisition of valuable intelligence, both medical and tactical. In evaluating the programs, Wilensky largely looked at these activities as failures from a medical standpoint. Wilensky generalized "There was virtually uniform agreement among physicians that single-visit, drop-in, unplanned, and uncoordinated MEDCAP visits were of negligible medical value, at best" [8].

Quoting the cautionary conclusion of Lt Col Peter B. Cramblet, MSC in his US Army War College thesis on medicine in low intensity conflict, "Exercises that accumulate impressive statistics for patients treated are a meaningless method of management by body count," a clear allusion to the failed strategy of "body count" for evaluating overall strategy in Vietnam [9]. Wilensky reported, "MAAG recognized that MEDCAP was not an example of the highest quality of medical service that the United States could offer the world. MEDCAP was never intended to be a purely medical effort, but primarily intended as psychological aid to combating VC infiltration. The author of the MAAG study thought it was possible that those opposed to the program never understood its underlying principles or stimulus." Wilensky continued, "MAAG felt that caring for civilians could be an opening wedge to create contact with the people of Vietnam, allowing other activities in the future" [10]. However, soldiers assigned to PSYOP, psychological operations, civil affairs, and Special Forces units are appropriately proud of their activities. While they all tell the stories of the MEDCAPS, DENTCAPS, VETCAPS, preventive meds actions, small building projects such as digging well and building schools, Mike Smith related a telling vignette that is applicable to today's activities in our GWOT. Mike Smith said, *"We dug wells and built schools, but mostly we defended the villages from acts of terrorism. The North Vietnamese and the Viet Cong regularly used torture, rape, and murder to intimidate the villagers. They came in the night to hold indoc-*

trination classes for the villagers. These indoctrination classes often consisted of gathering all the citizens of a village to watch the public gang rape of the wives and daughters of unfriendly civic leaders. Sometimes the civic leaders were beheaded. Sometimes, the villages were burned. Sometimes the young men were kidnapped and conscripted into the Communist army. Sometimes the right hand of every man, woman, and child was chopped off. Middle Eastern terrorists have nothing on Communist terrorists" [11].

As admitted by Wilensky, then Col Spurgeon Neel described a program where US military hospital admitted selected Vietnamese civilians for "high impact" surgical procedures. In this program, Vietnamese civilians, primarily children, with serious defects, deformities, and functional impairments were admitted for corrective surgery. These surgical procedures, performed on an elective basis, did not interfere with the primary mission of hospital, and in fact, enhanced the morale and capabilities of army surgeons by providing humanitarian and professional opportunities between the demands of peak combat casualty load. The psychological impact on the inhabitant of the village to which the restored patient returned was tremendous" [8, 12].

Several ophthalmologists documented the success of "winning the hearts and minds" type of eye care performed by several active duty ophthalmologists during their year-long tour of duty in Vietnam [13]. The opportunity to provide eye care for injured and diseased Vietnamese civilians came from a variety of sources. Some Army ophthalmologists traveled to Vietnamese hospitals, which often had religious affiliations, while others had patients brought to their US Army hospital by charitable religious organizations or by US Army MEDCAP members whose responsibility it was to do so. Vietnamese patients requiring surgery were admitted to the hospital and given the same care as that received by US service members. Under the rubric of civic action might also be included efforts to improve the instruction of Vietnamese medical students in ophthalmology. Because of the shortage of trained Vietnamese ophthalmologists and the ocular problems rampant in the civilian and military populations, and entirely on their own volition, most US Army ophthalmologists provided medical and surgical care for Vietnamese civilians and military in both Vietnamese and US Army hospitals. Leavitt, when not caring for US patients, found it possible to participate actively in the MEDCAP (medical civic action program) and to help establish a small eye clinic at the Holy Family Hospital in downtown Quin Nhon. Leavitt saw 20–40 patients a day, most of them with severely scarred corneas and major eyelid abnormalities. A number of cataract extractions were performed on lepers who were provided with +10 spherical corrections. It was common to see patients who had suffered corneal ulceration and perforation with resultant adherent leukoma, perhaps indicating the interactive effects of vitamin A deficiency and measles. Anderson provided similar care for Vietnamese patients at the 24th Evacuation Hospital and at the Catholic Hospital in Ho Nai with Brother Bernard Samuel, a registered nurse and skilled eye surgeon. Anderson saw a small truckload of civilian outpatients on a weekly basis, brought to the 24th Evac by Brother Bernard, and weekly did surgery for him, mostly cataracts. Also, knowing that no trained ophthalmologists would be available after his departure, he taught Brother Bernard to do cataract surgery, acting as his assistant during his last weeks. Anderson also served as consultant to a civilian hospital in Bien Hoa, which was served by Australian civilian doctors. Hornblass performed between 75 and 100 cataract or glaucoma operations on Vietnamese and Montagnard patients and provided weekly consultations at the leprosy center. He published a study of 50 leprosy patients and provided ophthalmologic training to three physicians and one ophthalmologist of the army of the Republic of Vietnam. He taught a 26-year-old Montagnard man to do cataract and ophthalmic plastic surgery. Aphakic lenses were purchased in Hong Kong through the efforts of Hornblass with funds provided by American service members who purchased craftwork from the Montagnard people. LaPiana treated Vietnamese civilians and military at the 3rd Field Hospital, Cong Hoa, and Ho Nai hospitals. When the army patient load permitted, he admitted indigent

Vietnamese civilians to the 3rd Field Hospital for cataract, glaucoma, oculoplastic, orbital, corneal, and extraoccular muscle surgery. These patients were brought to him by various religious organizations and US Army MEDCAP personnel. He lectured to, assisted in surgery Vietnamese army ophthalmologists at the Cong Hoa hospital, and together with Bernard Hodgkinson, MD, taught ophthalmology to Vietnamese medical students. Ehmer described the performance of the following 303 procedures on Vietnamese civilians: (1) cataract extraction 136, (2) pterygia removal 77, (3) cicatricial entropion repair 52, (4) tarsorrhaphy 20, (5) filtering procedures, 8 (6) peripheral iridetcomy 7, (7) involutional spastic entropion repair 3.

The provision of eye care to indigent native peoples was in Vietnam a major source of satisfaction to the care providers as well as an opportunity for them to expand their skills. One way it differs markedly from care provided to US personnel was the absence, much if not most of the time, of any ability to refer indigenous patients for further care. This fact justifies efforts to attempt to provide a level of care for such patients beyond that usually provided US casualties who will be evacuated to general hospitals. Thus, a complex lid reconstruction (or an orbital tumor resection) might be attempted by a general ophthalmologist, while in the United States such cases would be referred to subspecialists in a general hospital. This reality should be imparted to Army ophthalmologists before deployment. It may be necessary for the ophthalmologist to train nonmedical personnel in the performance of selected ophthalmic surgical procedures so that such individuals may continue to provide care for members of their community after US personnel have been withdrawn. The proven capabilities of military medics, especially SOF (Special Operations Forces) medics and corpsmen provide further justification for this [13].

In his US Army War College thesis "Medical Civic Action Programs (MEDCAPS) and medical readiness training exercises (MEDRETES) as instruments of foreign policy," Col (Dr) El Ray Jenkins noted that the Army Medical Corps had conducted humanitarian and civic action programs since the 1960s [14], initially for the Vietnamese military and later for Vietnamese civilians. Jenkins suggested the "the primary objective was to win the 'hearts and minds' of the people," concluding that medical care for civilian populations could play an important role in counteringinsurgency. It was his thesis that humanitarian and civic action programs can play a very important role in countering insurgencies and in nation building. Jenkins concluded that the "overall consensus was that the program was a success. Only after the war did opinions change" [14]. The issue of medical operations in low intensity conflict was evaluated quite nicely by then Lt Col Cramblet, MSc, in his 1991 US Army War College thesis, "U.S. Medical Imperatives for Low Intensity Conflict." Considering post-Vietnam actions conducted in various low intensity environments to include Beirut, Honduras, El Salvador, and Panama, Cramblet summarized what he felt were the key elements of successful medical operations in low intensity conflict based on doctrine and then recent medical experience. He noted that extensive MEDCAPs in Honduras starting in 1983 with joint exercise AHUAS TARA II (Big Pine) were described by the 7th Special Forces Group (Airborne) Surgeon as "totally independent uncoordinated civic action/humanitarian assistance activities" [9].

After the initial "mission accomplished" end of initial combat operations, both in Afghanistan and Iraq, most follow-on military and nonmilitary aid was intended to promote stability objectives, largely according to US COIN doctrine. According to the US Army FM 3-07, *Stability Operations*, "the greatest threat to our national security comes not in the form of terrorism or ambitious powers, but from fragile states either unable or unwilling to provide for the most basic needs of their people." In addition, although the US military has been very good at defining stability operations, low intensity conflict, combat operations, etc., the description of "winning the hearts and minds" is more problematic. According to US Army's 2006 FM Field Manual 3-24, *Counterinsurgency*, "winning hearts and minds" is described through examples rather than specific definition: "Once the unit settles into the

AO (area of operations), its next task is to build trusted networks. This is the true meaning of the phrase 'hearts and minds,' which comprises two separate components. 'Hearts' means persuading people that their best interests are served by COIN success. 'Minds' means convincing them that the force can protect them and that resisting it is pointless. Note that neither concerns whether the people like the Soldiers and Marines. Calculated self-interest, not emotion is what counts. Over time, successful trusted networks grow like roots into the populace. They displace enemy networks, which forces enemies into the open, letting military forces seize the initiative and destroy the insurgents."

In his 2008 US Army War College thesis, "Strategic Medical Leadership in the Global War on Terrorism," Col (Dr) Dallas Homas concluded that "fundamental to winning the hearts and minds of the local populace in countries like Afghanistan and Iraq, and in regions like the Horn of Africa, is ensuring basic human needs" and included security and health among those such needs. Colonel Homas catalogues the well-intentioned work by coalition PRT (Provincial Reconstruction Teams), rebuilding schools and clinics destroyed by combat operations, not realizing that there were insufficient doctors, nurses, and teachers to staff them. Still, Homas does state that the "medical outreach to the native population has been shown to be an effective way of endearing the people to the presence of our combat forces." According to Homas, "countless anecdotes are told of weapons caches being revealed to U.S. forces after the unit's medic had effectively treated the village elder's daughter, his prize cow, or another tribal member." Homas continued, "regional security can also be enhanced through medicine. In one volatile area of Afghanistan, near the Pakistan border, a military vehicle was struck by an improvised explosive device (IED) in 2004. It was commonplace for the military compound in theater to conduct sick call for the local populace on a daily basis, partnering Afghan doctors with U.S. doctors, physician's assistants, and medics. Following the IED strike, the base commander canceled sick call for a period of time, perceiving the threat to his base as too great, given the recent attack. This decision was conveyed to the village elders. When they realized that allowing the Taliban to place the IED resulted indirectly in the loss of their healthcare, the villagers actively set about ensuring that no further IEDs were place. After a few weeks of such enhanced security, the military commander resumed the operation of the medical clinic to the delight of the local villagers. In this instance, the Afghan village had done exactly what the USARPAC Commanding General had defined as victory in the GWOT. They had come to value a societal institution – the local medical are – to such an extent that they denied sanctuary to previously active Taliban operatives" [15].

An extensive review, "*Winning Hearts and Minds? Examining the Relationship between Aid and Security in Afghanistan*," was published from the Feinstein International Center in January 2012. According to the authors, Paul Fishstein and Andrew Wilder, political factors such as the corrupt and predatory behavior of government actors most frequently cause people to oppose the government and to support violence against it, yet most stabilization initiatives have placed importance on the economic drives of conflict, focusing on poverty, unemployment, illiteracy, social services, and infrastructure. Evidence from the study has shown that the pressure to spend too much money too quickly is wasteful and undermines both security and development objectives. Also, since the primary objective of US aid to countries such as Afghanistan is to promote security, with priorities promoting funding for insecure areas, with Afghans living in more secure areas feeling penalized for being peaceful. Aid resources have been viewed first and foremost as a stabilization tool or a "weapons system." And although there is considerable evidence that development assistance in Afghanistan is a good in and of itself, major development gains (decreased infant and maternal mortality, dramatic increases in school enrolment, major improvements in roads and infrastructure, and greater connectivity through telecommunication networks) have often been underappreciated because they did not translate into tangible security gains [16].

Jamie A. Williamson argues that "using humanitarian aid to 'win hearts and minds' is a costly failure," doing more harm than good. The author contends that counterinsurgency operations, such as in Afghanistan and Iraq, short-term incentives, and concessions do not necessarily go hand-in-hand with long-term transformation strategy. Williamson suggested that "while military-administered aid projects might help at a tactical level by allowing the international forces some interaction with the local community, to gather 'atmospherics and intelligence', they have little long-term overall strategic effect." Williamson quoted Kicullen's blunt rationale: "In a counterinsurgency, the gratitude effect will last until the sun goes down and the insurgents show up and say, 'You're on our side, aren't you? Otherwise, we're going to kill you" [17].

The authors contend that WHAM ophthalmology activities were definitely successful on the tactical level in support of military operations in Afghanistan. As described in an earlier article by Enzenauer and Vavra, the Army National Guard has extensive capabilities that are impossible to attain in active duty units, largely because its citizen soldiers bring their civilian expertise in addition to the military specialties. Before leaving CONUS, Enzenauer suspected that he might be "recruited" to provide some level of eye care during his deployment as Battalion Surgeon with Colorado's National Guard Special Forces, so he had requested a Field Optometry Set (slit lamp, phoropter, chair, etc.) but this was denied since there was not an eye doctor assigned to a Special Forces (SF) battalion. When deploying to Afghanistan as the flight surgeon for a forward deployed Special Forces (SF) battalion, Enzenauer also provided basic ophthalmologic care in theater in the absence of an assigned ophthalmologist. The SF Battalion Preventive Medicine NCO, SSG Vavra, was also an experienced eye technician in civilian life, having served with Enzenauer at Fitzsimons Army Medical Center 1985–1992. During OEF2, Operation Enduring Freedom 2, there was not an assigned ophthalmologist at the Combat Support Hospital in Bagram. After initial combat operations were complete in OEF1, Afghanistan evolved into a "relatively" safe combat zone. So when the first Combat Support Hospital (CSH) rotated home, the replacement unit did not have an ophthalmologist. Because there was not an assigned ophthalmologist, there were no ophthalmic surgical sets. Through the generous support of two former military ophthalmologists, colleagues from the now closed Fitzsimons Army Medical Center in Colorado (Dr Will Waterhouse, a retina specialist, and Dr. Stu Farris, an oculoplastic specialist) supplied sutures, a microinstrument set, a Simcoe I/A (irrigation/aspiration) device, Wek® cell sponges, an indirect ophthalmoscope, and antibiotic eye drops and ointment. The CSH had a 20-year-old Wild® operating microscope without a focusing pedal. However, the hospital commander at the time was an old army colleague of Col Enzenauer from Fitzsimons Army Medical Center, in the early 1990s – so in the absence of an assigned ophthalmologist, he recruited him to assist with emergency eye care at his facility. Consequently, emergency eye surgery was provided for allied, coalition, and host nation individuals in accordance with the Rules of Engagement. So, this is how the Colorado Army National Guard "eye service" (or the Tennessee Volunteer Medical Center, since Enzenauer had deployed with Colorado SF from Chattanooga, TN) was equipped [18]. Emergency eye care was provided for two of their own ARNG SG soldiers, along with their Afghan interpreter, who were all injured from an improvised explosive device (IED) that was thrown into their vehicle. Also care was provided for many injured Afghan civilians who sustained eye injuries in conjunction with other trauma, most commonly a result of accidental explosions of 20-year-old Russian mines. Most eye emergencies involved injured Afghan children, typically an injured child who was nearby a fatally injured child who had set off a mine. Consistent with the WHAM mission, Enzenauer cared for several coalition soldiers who presented with noncombat-related ocular trauma, from bunji cords, along with a foreign ambassador serving in his embassy who had glaucoma and cataracts that had been treated before at the Moorfields Eye Hospital in London.

The SF soldiers were excellent sources of referrals, often finding children in need of ophthalmologic care in the villages near their Area Operating Base (AOB) when working with deployed Afghan National Army units. Most commonly, children presented many days after blinding eye injuries, both to the CSH and to military units stationed around the country. With these patients who were beyond hope for recovery of vision, evisceration or enucleation was performed both for cosmesis and to prevent sympathetic ophthalmia in the uninjured eye. Since a formal ophthalmologic surgical supply chain was not available, to maintain orbital volume surgeons ended up using a sterilized child's marble, obtained through donations by the battalion chaplain, MAJ Andy Meverden, who also served as the operating room photographer. Another very interesting case involved a child who was injured a week earlier when a mine exploded, resulting in the loss of a major amount of brow and lid tissue. Miraculously, the eye itself was intact but vision was threated because of cicatricial ectropion. Normally, a lid reconstruction would have been performed utilizing post-auricular skin grafts. However, as seen in the preoperative photo, there was extensive cranial burn injury from the blast. After appropriate consent from the boy's father, the hospital commander, a general surgeon himself, performed a circumcision, providing the ophthalmic surgeon with graft material to reconstruct the lid. The excised foreskin provided an excellent repair; a temporary tarsorraphy was placed, and removed 1 week later. Additionally, nonemergency elective removal of periocular and ocular tumors were possible. Enzenauer was often brought to areas around the country to evaluate kids with poor vision. Many times, working in remote areas of Afghanistan with their affiliated Afghan National Army unit, American SF A-teams would discover children with strabismus and, after talking with the village elders, would say "our doc can take care of that!" With the consent and coordination of the CSH commander (an airborne-ranger infantry officer before medical school), he was able to perform many elective strabismus surgeries at the CSH in Bagram, with the expert assistance of his preventive medicine NCO, and former military eye technician (MOS 91Y), the two having literally performed hundreds of eye surgeries two decades earlier together on active duty. Interestingly, he saw no girls with strabismus; only boys were brought for surgical correction of their ocular misalignment. Additionally, through the Public Affairs Officer, a connection was made with the Kabul Medical University, and the neighboring local charity eye hospital, the Noor Eye Hospital (Noor translates to enlightenment in the Dari language.) The Noor Eye Hospital is an NGO-sponsored facility (IAM, International Assistance Mission), with one US-trained ophthalmologist working with a team of 5–6 Afghan ophthalmologists. Their surgical care was largely restricted to cataract surgery. The Muslim holy day Friday was the one day in seven that was a "day off." Since the SF soldiers were supporting the Afghan National Army. The "eye surgery team" from the FOB, the forward operating base, traveled generally monthly to Noor Eye Hospital in Kabul to examine strabismus patients and perform eye muscle surgery with the Afghan eye surgeons (http://www.iam-afghanistan.org/what-we-do/eye-care/noor-eye-hospitals/noor-eye-hospitals). Enzenauer regularly gave lectures to the staff at the Noor Eye Hospital. Enzenauer actually completed a study of Vitamin A deficiency in Kabul with the help of the staff at the Noor [19]. On his second deployment, the first time to Iraq, he operated on a young teenaged female who had a consecutive exotropia after surgery for primary infantile esotropia as a young child, assisting his former Fitzsimons Army Medical Center intern from 1990 who went onto an ophthalmology residency at Brooke Army Medical Center and a cornea fellowship at Emory. The teen's father was an Iraqi national who was a valuable intelligence asset that was being "courted." Intelligence individuals had contacted then LTC (Dr) Anthony Johnson if he could "fix" strabismus. Johnson replied that strabismus is not really his specialty but added that his former chief and civilian strabismus expert was "in country" with the Army National Guard. When Enzenauer shook hands with the father prior to his daughter's surgery, he found that the Iraqi was missing three fingers

from one hand – the fingers amputated during Saddam Hussein-inspired torture.

The performance of elective WHAM "winning the hearts and minds" ophthalmologic surgery was very successful on a tactical level in the area where the Special Forces battalion was operating. When the unit went back repeatedly to a local orphanage on humanitarian mission, one of the leaders approached the chaplain wanting to tell him who "in the neighborhood" was Taliban. He immediately referred them to the intelligence section. After SF Medical Sergeants and the assigned Physician Assistants, both former 18 series soldiers, rendered care to local host nationals, most often children with burns or referred kids with eye problems to "their battalion surgeon" and eye doctor, the attacks on FOBs reduced dramatically. There is no question that elective eye surgery was an excellent method to build rapport and trust in keeping with the Special Forces "winning the hearts and minds" philosophy. And, in contrast with the CSH in Bagram, Special Forces teams lived among the Afghans, with most of the FOBs (forward operating bases) far away from Kabul [19].

As the NATO portion of the effort in Afghanistan matured, more and more European-based medicine appeared on the battlefield. Many NATO participating countries have "civilian based" medicine accompanying their military. This resulted in more "civilian-centric" and "national-centric" medicine in the allied effort in Afghanistan. At the same time, American military thought on MEDCAPS began to change. The American Army began to question if performing direct patient care was effective in the counterinsurgency mission [20]. This groundswell was led by a former Civil Affairs unit surgeon, Rob Malsby, in his Command and General Staff College thesis, but soon spread to the line officers of the Army too. In a groundbreaking article in *Military Review,* a Cavalry Squadron commander and his regimental surgeon promulgated a wide brush article on whether or not the current concepts in counterinsurgency medicine were having the desired effects or were just responsible for unintended consequents. It also touched on the contentious issue of intelligence collection through medicine, a hot button item for the Europeans. In the article's conclusion, the authors state:

See general, battalion and BCT medical forces should not attempt to provide diagnostic and curative medical care to civilians, except in emergencies or in situations in which U.S forces inadvertently caused the injury. Regardless of the commander's motives, using the illusion of healthcare to engage the local population risks causing medical harm to those he intends to help, and perhaps more significantly, risks making tactical errors that are likely to undermine counterinsurgency strategy. A commander can most effectively improve the health of civilians in his area of responsibility by treating the disease of insecurity rather than attempting to treat its symptoms. Safety, dispelling fears caused by insurgent propaganda, and increasing freedom of movement" [21].

The Special Operations Forces in Afghanistan countered this pessimistic big army view of WHAM with their development of MEDEXs or medical engagement exercises. These exercises focused on education and inclusion of local providers, thus both putting a "local face" on the effort and by not undermining the economic base of the local providers' sustainability. It also resulted in more sustainable medicine as what was taught to local providers damped down the tendency to abruptly raise local medicine to American, hence unsustainable, first-world standards. The US Air Forces Special Operations developed a good source for guidelines on a successful medical engagement strategy [22].

As Afghanistan winds down, both the US SOF and conventional forces are beginning to talk more about "phase zero." A new term to the lexicon, it consists of, "taking coordinated action in peacetime to affect the strategic environment" [23]. China does it well, but the United States currently does not. Clearly, WHAM and medical engagement will again be at the tip of the spear (Figs. 15.1, 15.2, 15.3, 15.4, 15.5, 15.6, 15.7, 15.8, 15.9, 15.10, 15.11, 15.12, 15.13, 15.14, 15.15, 15.16, 15.17, 15.18, 15.19, 15.20, 15.21, 15.22, 15.23, 15.24, 15.25, 15.26, 15.27, and 15.28).

Fig. 15.1 Dr. Leavitt at his CSH

Fig. 15.2 Dr. Leavitt examining a leprosarium patient

Fig. 15.3 Dr. Anderson and Brother Bernard Samuel

Fig. 15.4 Dr. Hornblass with Montagnard patients

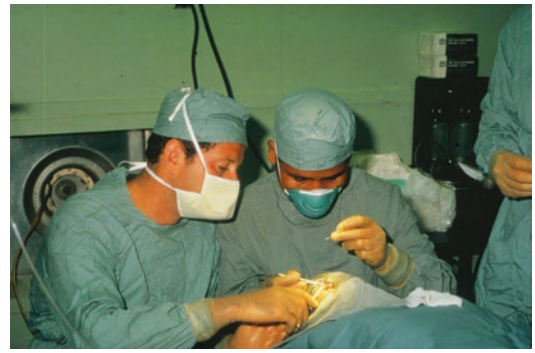

Fig. 15.5 Dr. Hornblass and a Montagnard student

Fig. 15.6 Dr. La Piana examining host nation patient

Fig. 15.7 Vietnamese patients who underwent sugery by Dr. La Piana at the 3rd Field Hospital

Fig. 15.10 Orbital tumor patient of Dr. Hornblass

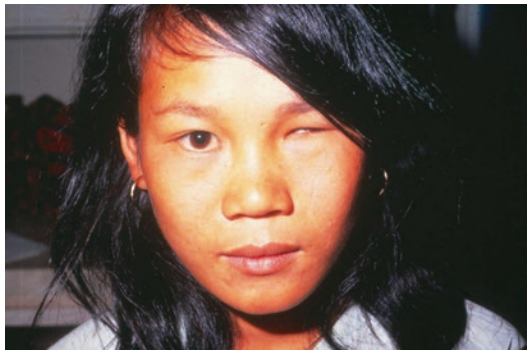

Fig. 15.8 Pre-operative oculoplastic patient of Dr. Hornblass

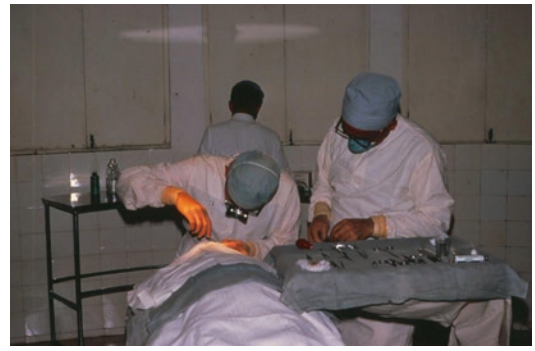

Fig. 15.11 Dr. La Piana performing surgery with Brother Bernard at Ho Nai Hospital

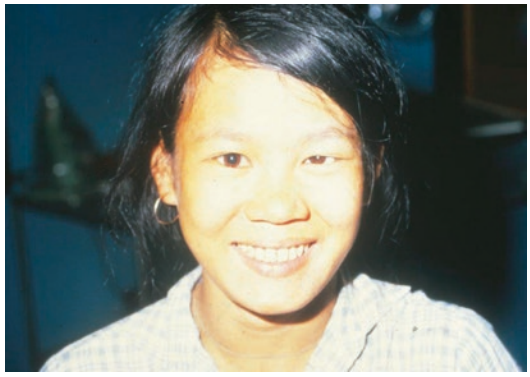

Fig. 15.9 Post-operative patient, Dr. Hornblass

Fig. 15.12 "Enzenauer Volunteer Medical Center," the 5/19th Battalion Medical Section, FOB 195, outside of Kabul, OEF2, 2002–2003

Fig. 15.13 The SF ODAs seen supervising security operations of the Afghan National Army for our local MEDCAPs (Medical Civic Action Programs) as part of some of the earliest confidence-building missions

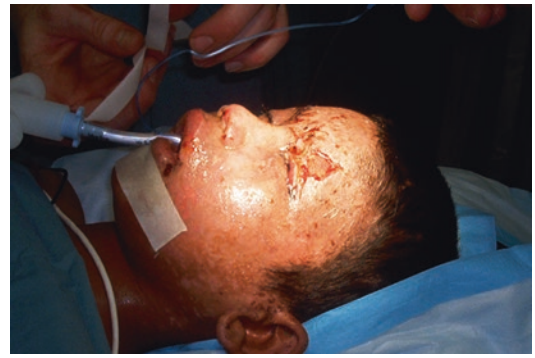

Fig. 15.16 Pre-op child with cicatricial ectropion with intact globe

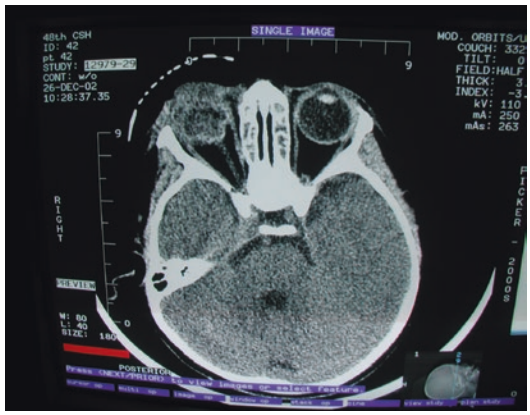

Fig. 15.14 CT scan documenting serious ocular disorganization without any other serious cranial abnormality

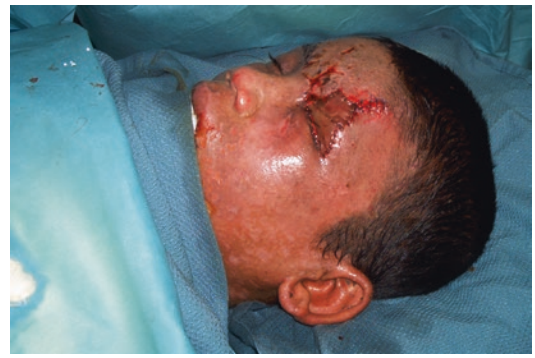

Fig. 15.17 Post-op child after foreskin used to reconstruct lid and brow

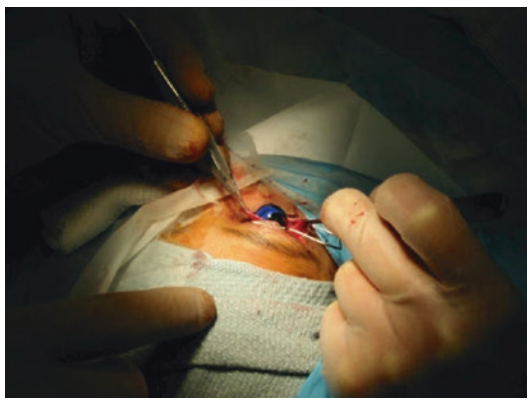

Fig. 15.15 A sterile marble being placed in the globe after evisceration

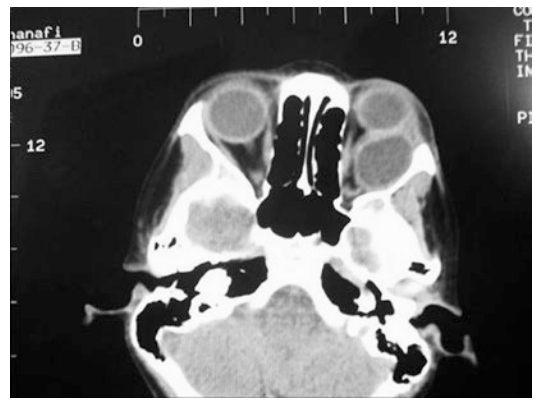

Fig. 15.18 CT scan reveals a large orbital dermoid that is threatening vision

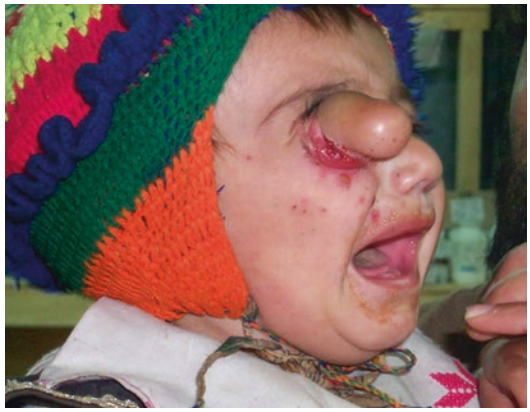

Fig. 15.19 A large tumor protruding out of a blind right eye

Fig. 15.20 Author Enzenauer examining blind child at the palace in downtown Kabul

Fig. 15.21 Author Enzenauer with SSG Vavra performing elective strabismus surgery at CSH

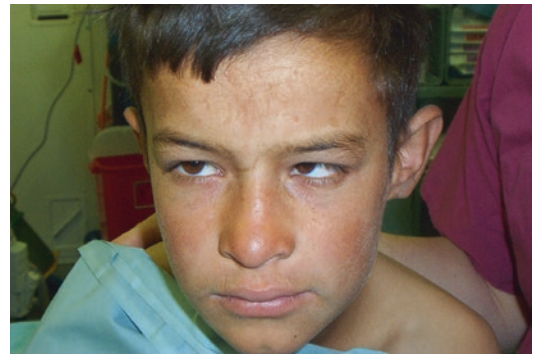

Fig. 15.22 Pre-op esotropia patient #1

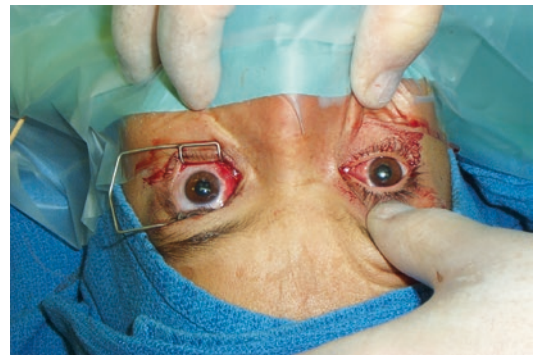

Fig. 15.23 Post-op esotropia patient #1

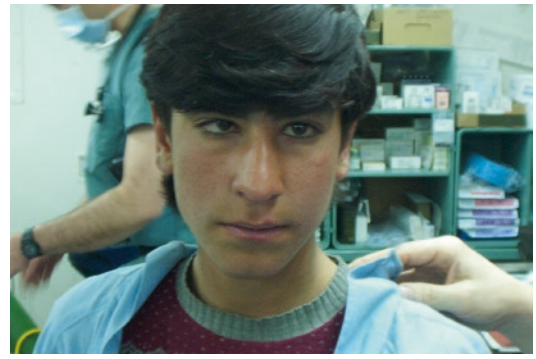

Fig. 15.24 Pre-op esotropia patient #2

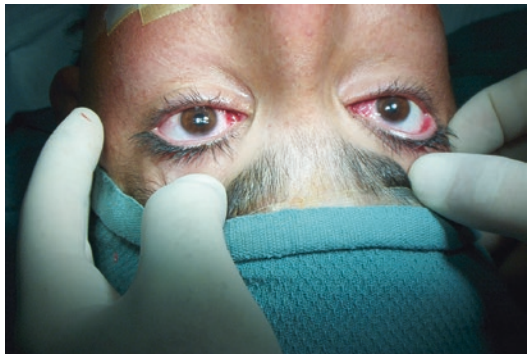

Fig. 15.25 Post-op esotropia patient #2

Fig. 15.26 Noor Eye Hospital operating theater, Kabul

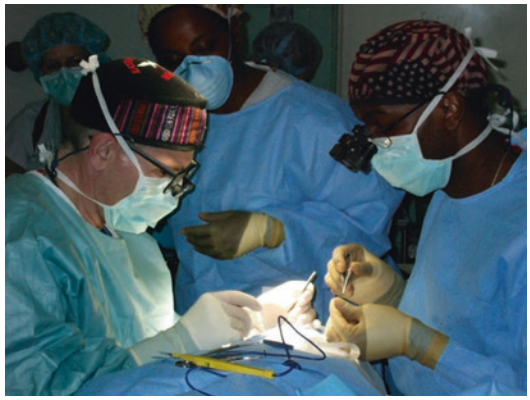

Fig. 15.27 Dr. Enzenauer assisting LTC Anthony Johnson is elective strabismus case in Iraq OIF2

Fig. 15.28 Happy father after "winning the hearts and minds" strabismus surgery on his son, Bagram CSH

References

1. Armstrong KK. Army Medical Department Support to Stability Operations. USAWC Strategy Research Project. Carlisle Barracks U.S. Army War College, February 28, 2007. 28pp. (AD-A469 380) http://www.dtic.mil/dtic/tr/fulltext/u2/a469380.pdf.
2. Headquarters, Joint Staff, Joint Publication 3-24, Counterinsurgency, 22 November 2013, Executive Summary, Commander's Overview, page ix.
3. Headquarters, Department of the Army, FM 3-24.2 (FM 90-8, FM 7-98), Tactics in Counterinsurgency, April 2009, Introduction, page ix.
4. Headquarters, Department of the Army, FM 3-24 MCWP 3-33.5, Insurgencies and Countering Insurgencies, May 2014, p 7-4.
5. Liddy L. The strategic corporal. Some requirements in training and education. Aust Army J. 2009; 11(2):139.
6. de Schweinitz GE. Ophthalmology (United States) and Greenwood A, Ophthalmology (American Expeditionary Forces) , in volume XI, surgery, part two, from the Medical Department of the United States Army in the world war. Washington, D.C.: Government Printing Office; 1924. p. 659–728.
7. Randolph ME. Ophthalmology Editor, in Ophthalmology and otolaryngology, from surgery in world war II, Medical Department, United States Army. Washington, D.C.: Office of the Surgeon General, Department of the Army; 1957. p. 1–378. Introduction, p. 1–25.
8. Wilensky RJ. Military medicine to win hearts and minds: aid to civilians in the Vietnam war. Lubbock: Texas Tech University Press; 2004. p. 78–100.

9. Cramblet PB. U.S. medical imperatives for low-intensity conflict. USAWC military studies program paper. Carlisle Barracks U.S. Army War College, April 5, 1991. 23pp. (AD-A236–817) published online: http://www.dtic.mil/dtic/tr/fulltext/u2/a236817.pdf.
10. Wilensky RJ. Military medicine to win hearts and minds: aid to civilians in the Vietnam war. Lubbock: Texas Tech University Press; 2004. p. 101–30.
11. SGM Herb Friedman (ret.). Winning hearts and minds. Published online in PSYOP. (http://www.psywarrior.com/PsyopHealth.html), 51 pages.
12. Neel S. The medical role in army stability operations. Mil Med. 1967;132:605–8. Published online: http://www.cdham.org/wp-content/uploads/2013/05/1967-neel-mso-article.pdf.
13. LaPiana FG, Hornblass A. Military ophthalmology in the Vietnam war. Doc Ophthalmol. 1997;93:29–48.
14. Jenkins ElRay. Medical Civic Action Programs (MEDCAPS) and Medical Readiness Training Exercises (MEDRETES) as Instruments of Foreign Policy. USAWC Strategy Research Project. Carlisle Barracks: U.S. Army War College, May 24, 1988. 75pp. (AD-A195 304) http://www.dtic.mil/dtic/tr/fulltext/u2/a195304.pdf.
15. Homas DW. Strategic medical leadership in the global war on terrorism. USAWC strategy research project. Carlisle Barracks: U.S. Army War College, March 21, 2008. 26pp. (AD-A479 005) http://www.dtic.mil/dtic/tr/fulltext/u2/a479005.pdf.
16. Paul F, Wilder A. Winning hearts and minds? Examining the relationship between aid and security in Afghanistan. Boston: Feinstein International Center, Tufts University; 2012. p. 82.
17. Williamson JA. Using humanitarian aid to 'win hearts and minds': a costly failure? International Review of the Red Cross. 2011;93(864):1035–61.
18. Enzenauer RW, Vavra DE. Combat ophthalmology when there isn't an assigned ophthalmologist. J Spec Oper Med. 2006;6(2):34–42.
19. Mihora LD, Jatla KK, Little I, Campbell M, Rahim A, Enzenauer RW. Vitamin A deficiency in Afghanistan. Eye Contact Lens. 2004;30(3):34–7.
20. Malsby RF III. Into which end does the thermometer go? Application of military medicine in counterinsurgency: does direct patient care by American Service Members Work?. Fort Leavenworth, Kan.: U.S. Army Command and General Staff College thesis; 2008.
21. Rice MS, Jones OJ. Medical operations in counterinsurgency warfare: desired effects and unintended consequences. Mil Rev. 2010;XC(3):47–57.
22. Thaler DE, et al. Technical report. Building partner health capacity with U.S. Military Forces. Enhancing AFSOC health engagement missions. Project Air Force: RAND, 2009.
23. McDonald SD, Jones B, Frazee JM. Phase zero. How China exploits it, why the United States dies not. Naval War Coll Rev. 2012;65(3):124–35.

Suggested Reading

Armstrong KK. Army medical department support to stability operations. USAWC strategy research project. Carlisle Barracks: U.S. Army War College, February 28, 2007. 28pp. (AD-A469 380) http://www.dtic.mil/dtic/tr/fulltext/u2/a469380.pdf.

Beath A, Christia F, Enikolopov R. Winning hearts and minds? Evidence from a field experiment in Afghanistan. April 13, 2012. MIT Political Research Paper No. 2011–14. Published online. \https://www.princeton.edu/~pcglobal/conferences/methods/papers/beath.pdf.

Benelli P, Donini A, Niland N. AFGHANISTAN: humanitarianism in uncertain times. Feinstein International Center, Tufts University, Boston, MA 02155, 51 pages, November 2012. Published online, http://fic.tufts.edu/assets/Afghan-uncertain-times.pdf.

Betson AP. A case study: tactics in counterinsurgency and a company during COIN operations. ARMOR. 2010 Sept-Oct: 32–36. Published online: http://fic.tufts.edu/assets/WinningHearts-in-Kenya.pdf, http://www.usma.edu/dmi/SiteAssets/SitePages/Current%20Faculty/FM%203-24.2%20and%20a%20Company%20In%20COIN.pdf.

Bradbury M, Kleinman M. Winning hearts and minds? Examining the relationship between aid and security in Kenya. Feinstein International Center, Tufts University, Boston, MA 02155, 78 pages, April 2010. Published online, http://fic.tufts.edu/assets/WinningHearts-in-Kenya.pdf.

Counterinsurgency – Selected Works. ARMOR September–October 2008, 78 pages. Published online: http://usarac.army.mil/cac2/AIWFC/COIN/repository/Armor_COIN_Selected_Works.pdf.

Cramblet PB. U.S. medical imperatives for low-intensity conflict. USAWC military studies program paper. Carlisle Barracks: U.S. Army War College, April 5, 1991. 23pp. (AD-A236–817) published online: http://www.dtic.mil/dtic/tr/fulltext/u2/a236817.pdf.

Department of Defense. 2007. Irregular Warfare (IW) Joint Operating Concept (JOC), version 1.0, Washington, DC: Office of the Secretary of Defense, 11 September.

Department of the Army, 2009. FM Field Manual 3–24.2 (FM 90–8, FM 7–98) tactics in counterinsurgency, Washington, DC: HQDA, 21 April.

Department of the Army, FM field manual 3–24/MCWP 3–33.5, insurgencies and countering insurgencies. Washington, DC, HQDA, 13 May.

Dickson E. 2009 A bright shining slogan – How "hearts and minds" came to be. Published on line. http://www.foreignpolicy.com/articles/2009/08/13/a_bright_shining_slogan.

Donini A. Humanitarian agenda 2015 Afghanistan country study. Feinstein International Center, Tufts University, Boston, MA 02155, 53 pages, June 2006. Published online, http://fic.tufts.edu/assets/Donini-Humanitarian-Agenda-2015-Afghanistan-Country-Study.pdf.

Donini A, Minear L, Smillie I, van Baarda T, Welch AC. Mapping the security environment: understanding the perceptions of local communities, peace support operations, and assistance agencies. Feinstein International Center, Tufts University, Boston, MA 02155, 98 pages, June 2005. Published online, http://fic.tufts.edu/assets/mapping_security.pdf.

Feinstein P. Winning hearts and minds? Examining the relationship between aid and security in Afghanistan's Balkh Province. Feinstein International Center, Tufts University, Boston, MA 02155, 66 pages, November 2010. Published online, http://fic.tufts.edu/assets/WinningHearts-Balkh-Province.pdf.

Fishstein P, Wilder A. Winning hearts and minds? Examining the relationship between aid and security in Afghanistan. Feinstein International Center, Tufts University, Boston, MA 02155, 84 pages, January 2012. Published online, http://fic.tufts.edu/assets/WinningHearts-final.pdf.

Fishtein P. BRIEFING NOTE: winning hearts and minds in Uruzgan Province. Feinstein International Center, Tufts University, Boston, MA 02155, 20 pages, August 2012. Published online, http://fic.tufts.edu/assets/Uruzan-Report.pdf.

Friedman JA. Manpower and counterinsurgency. Empirical foundations for theory and doctrine. Secur Stud. 2011;20(4):556–91. Published online: http://sites.dartmouth.edu/friedman/files/2014/07/Friedman-Manpower-and-Counterinsurgency.pdf.

Gompelman G. Winning hearts and minds? Examining the relationship between aid and security in Afghanistan's Faryab Province. Feinstein International Center, Tufts University, Boston, MA 02155, 73 pages, January 2011. Published online, http://fic.tufts.edu/assets/WinningHearts-Faryab.pdf.

Gordon S. Winning hearts and minds? Examining the relationship between aid and security in Afghanistan's Helmand Province. Feinstein International Center, Tufts University, Boston, MA 02155, 68 pages, April 2011. Published online, http://fic.tufts.edu/assets/WinningHearts-Helmand.pdf.

Hansen G. Taking sides or saving lives: Existential choices for the humanitarian enterprise in Iraq. Humanitarian Enterprise in Iraq. Humanitarian agenda 2015. Iraq country study. Feinstein International Center, Tufts University, Boston, MA 02155, 68 pages, June 2007. Published online, http://fic.tufts.edu/assets/HA2015-Iraq-Country-Study.pdf.

Hansen G, Independent Consultant. Coming to terms with the humanitarian imperative in Iraq. Humanitarian agenda 2015 briefing paper. Feinstein International Center, Tufts University, Boston, MA 02155, 25 pages, January 2007. Published online, http://fic.tufts.edu/assets/HA2015IraqBriefingPaper.pdf.

Hansen G, Independent Consultant. Taking sides or saving lives: existential choices for the humanitarian enterprise in Iraq. Humanitarian Enterprise in Iraq. Humanitarian agenda 2015. Iraq country study. Feinstein International Center, Tufts University, Boston, MA 02155, 68 pages, June 2007. Published online, http://fic.tufts.edu/assets/HA2015-Iraq-Country-Study.pdf.

Hansen G. Iraq: more challenges ahead for a fractured humanitarian enterprise. Briefing Paper. Humanitarian Agenda 2015. Feinstein International Center, Tufts University, Boston, MA 02155, 15 pages, November 2012. Published online, http://fic.tufts.edu/assets/hansen_iraq_BP.pdf.

Headquarters, Joint Staff, 2013. JP joint publication 3–24, counterinsurgency. Washington, DC: HQ Joint Staff, 22 November.

Hearts and Minds (Iraq). Wikipedia, the free encyclopedia. http://en.wikipedia.org/wiki/Hearts_and_minds_(Iraq).

Hearts and Minds (Vietnam). Wikipedia, the free encyclopedia. http://en.wikipedia.org/wiki/Hearts_and_Minds_(Vietnam).

Homas DW. Strategic medical leadership in the global war on terrorism. USAWC strategy research project. Carlisle Barracks: U.S. Army War College, March 21, 2008. 26pp. (AD-A479 005) http://www.dtic.mil/dtic/tr/fulltext/u2/a479005.pdf

James K. Winning hearts and minds during COIN campaigns: policy implications from Stathis Kalyvas' concepts of attitudinal and behavioural support. Small Wars Journal. 2012:10. Published online. http://smallwarsjournal.com/print/11356.

Jenkins ER. Medical Civic Action Programs (MEDCAPS) and Medical Readiness Training Exercises (MEDRETES) as instruments of foreign policy. USAWC strategy research project. Carlisle Barracks: U.S. Army War College, May 24, 1988. 75pp. (AD-A195 304) http://www.dtic.mil/dtic/tr/fulltext/u2/a195304.pdf.

Joint Warfighting Center, United States Forces Command. 2006. Irregular Warfare special study. Suffolk, VA, 4 August.

Lapidow CBL. Winning hearts and minds – Eagle style. engineer magazine; 2007 January–March, page 35–36. Published online: http://www.wood.army.mil/engrmag/PDFs%20Jan-Mar%2007/Lapidow1.pdf.

Mattox RM, Rodgers PS. 2007. Counterinsurgency in the 21st century: The foundation and implications of the

new U.S. Doctrine Master's Thesis. Naval Postgraduate School, Monterey, California. December, 121 pages. Published online: https://www.google.com/#q=Mattox+Raymond+M%2C+Peter+S.+Rodgers.+2007.+Counterinsurgency+in+the+21st+Century:+The+Foundation+and+Implications+of+the+New+U.S.+Doctrine+Master%E2%80%99s+Thesis.+Naval+Postgraduate+School%2C+Monterey%2C+California.+December%2C+121+pages.+ or www.dtic.mil/cgi-bin/GetTRDoc?AD=ADA475931.

Mazurana D, Stites E, Nojumi N. Human security and livelihoods of rural Afghans, 2002–2003. Feinstein International Center, Tufts University, Boston, MA 02155, 263 pages, June 2004. Published online, http://fic.tufts.edu/assets/Human+Security+and+Livelihoods+of+Rural+Afghans.pdf.

McClanahan Jr, Jack R. America's information war on terrorism: winning hearts and minds in the Muslim world. USAWC Strategic Research Project. US Army War College, Carlisle Barracks, PA 17013, 2002. Published online: http://www.iwar.org.uk/psyops/resources/hearts-minds/io-on-terrorism.pdf.

Neel S. The medical role in army stability operations. Mil Med. 1967;132:605–8. Published online: http://www.cdham.org/wp-content/uploads/2013/05/1967-neel-mso-article.pdf.

Pauly, Jr RJ, Redding RW. Part III: soft power. Chapter 14. Denying Terrorists Sanctuary through Civil Military Operations. 273–297. In: Forest James JF, editor. Countering Terrorism and Insurgency in the 21st Century: International Perspectives, Volumes 1–3, Volume 1 STRATEGIC AND TACTICAL CONSIDERATIONS. Praeger Security International, Westport, CT 06881, 2007. Available at: http://the-eye.eu/public/WorldTracker.org/Military/Terrorism%20and%20Counterterrorism/CounteringTerrorism.pdf.

Peck K. September 28, 2009. U.S. must win Afghan hearts and minds, commander says. Published on line. http://www.cnn.com/2009/POLITICS/09/28/afghanistan.obama/index.html?eref=onion.

Redding RW. 19th SF Group Utilizes MCA Missions to Train Afghan National Army Battalions. Special Warfare. 2005;17(3):22–27. Available at: https://www.dvidshub.net/publication/issues/8236.

Stephen B. Review symposium. The new U.S. Army/Marine Corps counterinsurgency field manual as political science and political praxis. Perspect Polit. 2008;6(2):347–60. Published online: http://www.apsanet.org/imgtest/popjune08counterinsurgency2.pdf?q=counterinsurgency-field.

The Infantry School. 1950. ST (Special Text) 31–20-1. Operations against Guerrilla Forces. Fort Benning, GA: September.

Thomas H. WHAM: winning hearts and minds in Afghanistan and elsewhere. JSOU report 12–1, February 2012.Joint Special Operations University, 7701 Tampa Point Blvd, MacDill AFB, FL 33621, 69 pages. Published online: http://jsou.socom.mil/JSOU%20Publications/12-1_WHAM_020112_final.PDF.

United States Army. 1951. FM field manual 31–20, operations against Guerilla Forces. Washington, DC: HQDA, 1 February.

United States Army. 1951. FM field manual 31–21, Organization and conduct of Guerilla Warfare. Washington, DC: HQDA, 5 October.

United States Army. 1955. FM field manual 31–20, US Army Special Forces Group (Airborne). Washington, DC: HQDA, 10 August.

United States Army. 1955. FM field manual 31–21, Guerrilla warfare. Washington, DC: HQDA, 23 March.

United States Army. 1958. FM field manual 31–21, Guerrilla Warfare and Special Forces Operations. Washington, DC: HQDA, 8 May.

United States Army. 1961. FM field manual 31–15, Operations against irregular forces. Washington, DC: HQDA, 31 May.

United States Army. 1961. FM field manual 31–22, US Army Counterinsurgency Forces. Washington, DC: HQDA, 31 May.

United States Army. 1963. FM field manual 31–16, Counterguerrilla Operations. Washington, DC: HQDA, 19 February.

United States Army. 1963. FM field manual 31–22, US Army Counterinsurgency Forces. Washington, DC: HQDA, November.

United States Army. 1965. FM Field Manual 31–20, Special Forces Operational Techniques. Washington, DC: HQDA, 30 December.

United States Army. 1965. FM field manual 31–21, Special Forces Operations. Washington, DC: HQDA, 5 June.

United States Army. 1965. FM field manual 31–73, advisor handbook for counterinsurgency. Washington, DC: HQDA, 23 April.

United States Army. 1967. FM field manual 37–73, advisor handbook for stability operations. Washington, DC: HQDA, 18 October.

United States Army. 1969. FM field manual 31–21, special forces operations. Washington, DC: HQDA, 14 February.

United States Army. 1971. FM field manual 31–20, special forces operational techniques. Washington, DC: HQDA, 12 February.

United States Army. 1972. FM field manual 31–23, Stability Operations US Army Doctrine. Washington, DC: HQDA, 2 October.

United States Army. 1978. ST special text 31–201, Special Forces Operations. Washington, DC: John F. Kennedy Special Warfare Center, November.

United States Army. 1986. FM field manual 90–8, Counterguerrilla operations. Washington, DC: HQDA, 29 August.

United States Army. 1990. FM field manual 100–20/ AFP 3–20, Military operations in low-intensity conflict. Washington, DC: HQDA, 5 December.

United States Army. 1992. FM field manual 7–98, Operations in low-intensity conflict. Washington, DC: HQDA, 19 October.

United States Army. 2001. FM Field Manual 3–05.201 Vol 1, Special Forces Unconventional Warfare Operations. Washington, DC: HQDA, 1 November.

United States Army. 2003. FM 3–07 (FM 100–20), stability operations and support operations. Washington, DC: HQDA, 20 February.

United States Army. 2006. FM field manual 3–24/MCWP 3–33.5, Counterinsurgency. Washington, DC: HQDA, 15 December.

United States Army. 2008. FM field manual 3–05.130, Army Special Operations Forces. Unconventional Warfare. Washington, DC: HQDA, 30 September.

War Department. 1941. FM basic field manual 31–20, Jungle Warfare. Washington, DC: US Government Printing Office, 15 December.

Williams K. The other side of COIN: counterinsurgency and community policing. Interface: A Journal for and About Social Movements. 2011;3(1):81–117. Published online: https://www.indybay.org/uploads/2013/12/10/the_other_side_of_the_coin_-_counterinsu_-_kristian_williams.pdf or http://www.interfacejournal.net/wordpress/wp-content/uploads/2011/05/Interface-3-1-Williams.pdf.

Winning hearts and minds. Wikipedia, the free encyclopedia. http://en.wikipedia.org/wiki/Winning_hearts_and_minds.

Zalewski P. June 26, 2014. Militants. ISIL: Winning Hearts and Minds, the Jihadist Way. 8 pages. Published online. http://www.businessweek.com/articles/2014-06-26/isils-jihadist-leaders-try-to-show-iraqis-they-can-govern.

Eye Armor

16

Thomas P. Ward and Francis G. La Piana

"If protection of the eyes of combat soldiers were simple affair, that protection would have been provided long ago."

– J. Fair, 1952 [1]

Introduction

Attempts to protect the soldier's body in armor have been made at least since the fifth millennium BC but attempts to protect the eyes only gained momentum in the past century [1, 2]. Up until the late twentieth century, the great majority of emmetropic American infantry men, the soldiers most at risk, entered combat with their eyes as exposed to the hazards of war as were the eyes of the first combatants. This chapter is an account of the multi-decade effort to develop eye protection for the American infantry man, an effort that finally resulted in effective eye armor that was accepted by the soldier in combat [3].

It is necessary to define *eye armor* as the term is used in this chapter because the eye is vulnerable to many threats, but protection against only some of them is necessary and possible. The major threat to the eye of the infantryman in combat is the small missile, as has been true since World War I [4]. *Eye armor* is defined primarily, though not exclusively, as that component of personal body armor that can protect the eyes of the infantryman from such a threat.

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Eye Injuries in War

Injuries to the eye and its adnexal structures are of increasing significance in war. The incidence of eye injuries sustained by US forces has increased 18-fold since the US Civil War, reaching 13% in the ground phase of Desert Storm [5]. In Operation Iraqi Freedom and Operation Enduring Freedom, 15.8% of all medical evacuations from Iraq and Afghanistan were for eye-related problems [6]. Making these figures even more ominous is the finding that 20–50% of ocular injuries are penetrating or perforating globe injuries and up to 28% are bilateral [7, 8]. Eye injuries are a common occurrence on the modern battlefield.

Not only are ocular injuries common in combat, they are also devastating. A soldier who sustained a penetrating wound of the globe in combat in Vietnam had a 50% chance of losing the eye no matter how prompt and expert the care [7]. Recent data from Operations Iraqi Freedom and Enduring Freedom reveal that close to a third of service members that experienced an ocular trauma became legally blind [9]. These figures should be compared to the dramatic decrease in the percentage of wounded dying from their wounds (from 14.1% in the US Civil War to 4.5% in World War II, 2.5% in the Korean War, and 2.6% in the Vietnam War) [3, 10]. Only 25% of the Vietnam eye casualties could return to active

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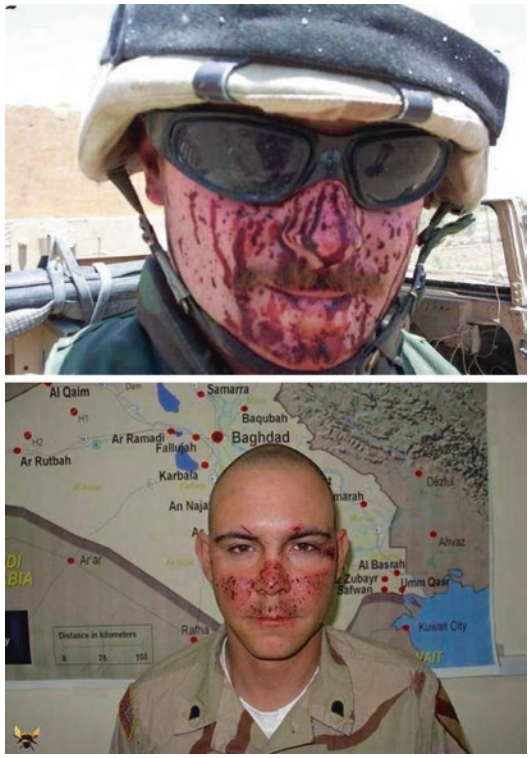

Fig. 16.1 An example of how modern body armor protects the head and neck from multiple fragments. Note how the forehead was protected by the helmet and the eyes and orbit by the eye armor

duty, while 83% of all surviving wounded could do so [11, 12]. The Wound Data and Munitions Effectiveness in Vietnam (WDMEV) team determined that 7.4% of interviewed casualties report reported "eye disability" after wounding [13]. Of these eye casualties, 79% were partially disabled and 21% completely disabled (at least temporarily).

The cost to society of eye injuries (both combat-related and during peacetime) is significant, both monetarily and medically. For example, a 20-year-old E-4 (specialist) who loses one eye in the line of duty will receive at least $580,000 over his expected lifetime, and an O–5 (Lieutenant Colonel) with 18 years of service will receive at least $1,500,000. Fortunately, the great majority of these accidents can be prevented [14]. This fact has resulted in the general requirement of the American National Standards Institute (ANSI) Z-87.1-1979 standard that "...eye protection shall be required in hazardous environments where there is a reasonable probability that injuries can be prevented by the use of such protection" [15]. It would seem appropriate that the same concern about eye injuries in the civilian workplace should exist for the soldier in combat.

Despite the obvious concern for ocular injuries on the battlefield, it must not be forgotten that soldiers are at risk for eye trauma even during peacetime. Tarabishy in 1983 reported that 40% (75 of 157) of injuries sustained by soldiers from four types of automatic weapons over a six-year period in peacetime were to the eye [16]. McMarlin and Connelly reported that 5% of injuries seen in an Army field hospital during a military training exercise were to the eyes [17].

Elements in Eye Armor Development

The development of eye armor was begun during World War I but did not reach fruition until just prior to the Gulf War, in large part because of the complexity of the task. Five elements must be considered: (1) the tasks of the infantryman; (2) the ocular threats; (3) the mind-sets of those to be protected and of those in positions of leadership; (4) the materials available to provide protection; and (5) available funds to support the costs of development, testing, modification, provision, maintenance, and replacement of eye armor.

The missions of an infantryman in combat can be reduced to firing his weapon or weapons, moving, identifying friend or foe, estimating range, communicating, and surviving. His eyes are his primary fire control mechanism and are important in maneuvering (as he is often the hunted as well as the hunter), and in communicating (a significant amount of which is done primarily with the eyes). The sine qua non for eye armor for the infantryman is that it must not only protect his eyes against several threats but must neither interfere with his ability to accomplish his missions nor with his chances of surviving them unharmed. Because the infantryman trains

and fights under the most rugged conditions, equipment provided him must be simple and very rugged. It must also be compatible with his equipment (e.g., helmet and weapon). Eye protection suitable for a pilot who fights seated and protected by his aircraft canopy may not serve for the infantryman who often must run, jump, and hit the ground hard and often. The detection of movement in the periphery of his visual field is of such great importance to the infantryman, correlating directly with his chances of survival, that he will reject any eye armor that interferes with his peripheral vision. This fact has been appreciated for at least 100 years – "...the fighting man must keep his whole visual acuteness, or at least have it but slightly modified by the protecting apparatus placed before the cornea; the visual field must not be manifestly narrowed" [4].

In the twentieth century, body armor (except for the helmet) has been worn mostly by those on the defensive [18]. If eyes are unprotected, soldiers on the defensive suffer more eye injuries than do those on the offensive [19]. Since the head and neck region of the soldier are the "locus of the major sensory equipment in the human ... continuous appraisal of his situation vis-a-vis the enemy forces the foot-soldier to expose his head more often than any other part of his body" [3]. Even taking this into account, an infantryman's eyes are injured at a frequency at least ten times higher than might be expected based on target size alone [20]. Eye injuries, furthermore, are always important; a small corneal abrasion can completely incapacitate a soldier in combat and penetrating injuries of the globe require medical evacuation.

In wartime, the major ocular threat is from fragments generated by detonating munitions, and we must expect that laser weapons will also be employed against our soldiers' eyes in any future conflicts. Eye-hazardous laser range finders and target designators are widely deployed now. The problem of protecting the eye against even a few wavelengths in such a way as not to impair the soldier's performance is a monumental task. The advent of the frequency-agile laser on the battlefield will only increase the problem [21]. Other significant immediate or potential threats to the eye are fragments from improved conventional munitions, flechettes (dart-like missiles released from artillery projectiles), ultraviolet light, flash from nuclear weapon detonation, sunlight, wind, dust, microwaves, particle beams, blast, heat, and poison gases. There is no way to protect against all of the threats all of the time, but it is now possible to protect against the small missile, the ultraviolet light, and blunt-force threats very well, and also against some of the eye-hazardous laser wavelengths. An analysis of ocular injuries to American servicemen in Vietnam estimated that the wearing of 2 mm polycarbonate eye protection would have prevented fully 39% of all ocular injuries [22].

The element in eye armor development that has been least appreciated is the mind-sets of both those who need protection and those who lead the Army. The complexity of the objective has frequently been ignored and the infantryman has often been regarded as just another industrial worker needing eye protection. In fact, the infantryman is usually young, emmetropic, unsophisticated, skeptical, denial-practicing, and body-image conscious, with a variety of highly dangerous tasks to perform (most of which require unimpeded vision) and burdened already with much personnel equipment. He tends to regard ametropia for what it is, an eye abnormality. He is likely to reject eye protection that resembles ordinary spectacles, both because of the implications of wearing it and the interference with his field of vision produced by the spectacle frame. Only three of the 92 American soldiers treated for ocular complaints at one combat support hospital in the first Gulf War were wearing their eye protection at the time [5]. It is important, therefore, not only to provide the infantryman with eye protection that provides a nearly unimpeded field of vision, but also to term it "eye armor" rather than "goggles" or "spectacles."

The mind-sets of those in senior positions are also of critical importance. Senior officers have often regarded eye injuries as being of little overall consequence and not preventable. The threat of injury to the infantryman's eyes has been

in part consciously and in part unconsciously denied because to recognize it would saddle the Army with a major additional task that in the past could not be accomplished. Certain groups of combatants have, however, been judged to need eye protection (e.g., aviators, tankers), reflecting the elitist division between cavalry and infantry known since antiquity. Sometimes it is the developers of eye armor who have failed to involve the user of the armor in its planning and development [23].

Materials available for the production of soldier-acceptable eye armor have been readily available for only a relatively short period of time. Eye armor development was retarded by the belief that the generation of secondary missiles by shattered glass lenses made their use for the protection of emmetropes unwise. The plastic lens, CR-39, was easily scratched, and neither glass nor plastic could be formed in a configuration that would protect the temporal portion of the globe without obstructing peripheral vision. The development of injection-moldable, optical-grade polycarbonate and scratch-resistant coatings has obviated all of these problems.

Fortunately for the US soldier, Army leadership has made available the monies required for the development, testing, and initial procurement of eye armor. Polycarbonate is intrinsically inexpensive and the cost to the US taxpayer for the infantryman's eye protection is far less than the cost of his boots. The elements that have been of greatest importance in the successful development of eye armor are the availability of injection-moldable polycarbonate, the decision of the Infantry School to make eye armor a requirement for the infantryman, and the fear of laser weapons.

The History of Eye Armor Development in America

Pre-Colombian Period to World War I

The Incan and Aztec warriors of pre-Colombian America wore quilted cotton jackets and padded helmets that did not incorporate eye protection [24]. The Colonial period saw the gradual abandonment of the metal body armor that the earliest settlers had brought with them from Europe because it was "...too burdensome for the long treks and rapid movements of woodland warfare" [25] despite its effectiveness against Indian arrows [24]. "Soft" armor of buckram (a stiff armor of cotton or linen and silk covered with leather), fustian (a type of cotton or linen fabric), or canvas was also used by the colonists but was discarded because it was hot and uncomfortable [26]. Though eye protection for the helmet wearer was attempted in the fifteenth century by means of "metal-rimmed protective lenses of glass ... hinged to drop over the eyes" [27], such eye protection was not present on helmets worn in the New World. Some of the Spanish infantrymen who accompanied DeSoto wore a type of helmet called a salade or sallet, some of which bore a hinged visor, and others themselves covered the face, in which case vision was provided for by means of a slot (ocularium) [25]. These partial eye protective devices were abandoned in part because the limitation of visual field they produced prevented the effective handling of pistols.

In the American Revolutionary War and the War of 1812, the cavalry continued to wear leather helmets and a few combat engineers wore steel breastplates [18, 24]. Breastplates were also worn in the American Civil War by combatants of both sides, although they were never formally authorized [28]. The Indian and Spanish-American Wars were fought apparently without body armor of any kind, though "push-shields" were considered for use in the latter [18]. By the onset of World War I, the use of body armor was regarded as "dead as Queen Anne" [26].

World War I and the Interwar Period (1914–1940)

Although all belligerent nations embarked on World War I providing little if any body armor for their infantrymen, almost all (including the United States) made efforts during that war to develop and distribute armor, including eye armor. The head was protected first, largely through the efforts of General Adrian of the French army, but

ophthalmologists soon attempted to stimulate and assist in efforts "...to try and realize for the eye sockets what has been obtained for the skull" [4, 18]. Unfortunately, no acceptable eye armor could be developed and development of eye armor for the infantryman practically stopped at war's end.

The major impetus for the interest in the development of body armor early in World War I was the employment by all armies of munitions generating a myriad of small fragments upon detonation, the extensive use of the machine gun, and the rapid replacement of a war of maneuver with a war of position ("trench warfare"), which made many combatants especially vulnerable to small fragment injuries. The trench war became a war of artillery and over half the casualties were caused by shellfire [29]. The static war of the trenches led to a peak of 8% of injuries being eye injuries, and 10% of all patients seen in base hospitals required eye examinations and treatment [10, 30, 31]. Three-quarters of the casualties were due to missiles of low velocity, less than 1000 ft/s [26]. A British 1917 attempt at eye armor was based upon a French automobile driver's goggle. Because of the inherent visual field limitations of these "lunettes," however, they would not have to be worn by soldiers, except when the wearer was "... under bombardment or menaced by bullets" [32].

Senior American Army ophthalmologists, such as Wilmer and Greenwood [33], were familiar with the various types of eye armor developed by our allies and with their deficiencies. Wilmer, at the request of the Ordnance Department, had developed an eye shield of Hadfield (manganese) steel with a single horizontal stenopeic slit and a circular opening below to permit a view of the ground. The idea for the shield came to Wilmer from the "single slotted eye shield which is used against snow blindness by the Indians of our northwest" [18]. Greenwood devised an "eye shield" with two stenopeic slits, one vertical and one horizontal, but concluded that Wilmer's shield, designed to be compatible with the standard British helmet, was superior [30]. The US Army ordered 30,000 of Wilmer's shield but they were rejected by the headquarters of the American Expeditionary Force because they were "not readily kept in position" [18]. This unfortunate result mirrored the fate of all body armor (except the helmet).

Although visors of different types were tested on experimental helmets of many different designs, all visors were rejected and the helmets that became standard at the outset of World War II made no provision for eye protection. In fact, many line officers in positions of authority during this period of time believed that eye protection would "spoil the image of the soldier," who was apparently expected to be farsighted in every sense of the word (Lowrey, personal communication, 1979). Efforts did continue to improve flying goggles for the Army and Navy aviators, for "it had long been realized that the task of flying was more dependent on vision than on any other of man's senses" [34]. It is interesting, though not surprising, that many of the issues dealt with by developers of better eye protection for military aviators in the 1920s and 1930s (field of vision, peripheral protection) are the same issues dealt with in the 1970s and 1980s for the infantryman.

World War II and the Interwar Period (1941–1949)

In contrast to the many efforts made in World War I, relatively few such efforts were made toward eye protection in World War II. Those in senior positions considered the incidence of eye injuries to be too low to necessitate eye armor development. Military planning in the pre-World War II period posited a war of movement, of maneuver, to obviate a recurrence of the static trench warfare of World War I. Body armor (except for the helmet) was believed to hinder the infantryman so much as to be ill advised [23]. In short, it was decided not to "sacrifice freedom of body movement for protection" [35]. Hence, although the prevention of industrial eye injuries was well advanced, the United States entered World War II with no eye armor for its infantrymen [36, 37].

Eye injuries were again very significant [20]. As had been true in World War I, the devastating effects of miniscule fragments upon soldiers' eyes prompted attempts at eye armor develop-

ment. Town [38] described a "metal eye protector," then in use by Soviet Union forces, which weighed 5 oz and provided for vision through crossed stenopeic slits. Stieren [39] described a "metal safety and glare goggle" of aluminum (reminiscent of the British World War I eye armor). An eyeshield of cellulose acetate was provided for members of the Chemical Corps for wear when they were in the vicinity of toxic gases, and some soldiers employed this eyeshield as a dust protector. A sun-wind-dust goggle, M1944, bearing 1 mm cellulose acetate lenses was provided to tankers and certain vehicle operators, but its size and shape made it unsuitable for use by foot soldiers. Toward the end of the war a metal eye shield, T45, was developed for engineers engaged in mine clearance [23, 40]. It was composed of a plate of manganese steel bearing vision slits (similar to those of the World War I British eye armor) mounted on a rubber sun-wind-dust goggle frame and weighed 7 oz.

US Army Air Force aviators wore several types of eye protection and different types of sunglasses, but the restriction of visual field was a major problem [34, 41]. The US Navy considered a visor for the standard M1 helmet to protect the face, but it was not fielded. Ironically, but not surprisingly, "industrial type" eye protection was provided to some soldiers performing equipment maintenance, and successful efforts were made by the US Armed Forces to protect the eyes of those working in defense industries [42, 43]. The glass spectacles worn by ametropic soldiers were not case-hardened and secondary missile injuries occurred with enough frequency to stimulate a recommendation that increased protection be provided the ametrope: "Ordinary spectacles should be made of armor plate or shatter-proof glass" [20]. Body armor, especially in the form of thoraco-abdominal protection, was investigated for infantry. "Flak suits" were developed for and extensively and effectively used by US Army Air Force flying personnel. Eye protection for these airmen was nonetheless suboptimal and many eye injuries occurred [23].

The British did make efforts to develop eye protection for the infantry. Cruise [44], who had developed a form of helmet-mounted eye armor termed the "chainmail veil" in World War I, had continued to work on such a protective device in the interwar period. In 1940, he advocated a helmet-attached perforated visor of 22-gauge duraluminum that could, if necessary, be adjusted over spectacles. The visor "acted as a multiple stenopeic disk, and in that way vision would be improved for the people with refractive errors without their glasses" [45]. The visors used by knights in the Middle Ages were also believed to correct refractive errors in a similar fashion. By 1941, three types of eye armor had been evaluated by the British military: (1) a perforated metal visor of the Cruise type; (2) slotted and round holed metal visors; and (3) methyl methacrylate and cellulose acetate plastic visors and goggles [46]. Cellulose acetate, 2 or 3 mm thick, was found superior to methyl methacrylate on impact resistance evaluation. The latter's proclivity to spall was to cost some airmen their sight during the war. The scratchability of the plastic was identified as a serious problem. Despite the efforts made to develop and field eye armor, British soldiers were provided no protection to the eye beyond cellophane anti-gas shields similar to the cellulose acetate shield provided US ground forces.

The Korean War and the Interwar Period (1950–1962)

The Korean War evolved from a war of maneuver to a war of position, and the resulting eye injuries again stimulated US Army ophthalmologists to attempt to enhance eye protection. King [47], a US Army ophthalmologist, called for the provision of case-hardened lenses to ametropic combat arms soldiers and considered an eye shield that could be attached to the helmet. He recommended the testing of plastic lenses in frontline companies and stressed the importance of gauging the soldier's acceptance or rejection of eye armor [48].

Freed, the inventor of the metal device described by Town [38], attempted to interest the US Army in it without success. Fair [49] made

the major eye armor development effort by advocating a "spectacle-type goggle with tempered glass lenses and side shields." He noted that "the only real problems foreseen are making the goggles acceptable to the soldier who has never before worn spectacles and providing lenses for the soldier with a significant refractive error" [49]. Unfortunately for thousands of US soldiers, accomplishment of these objectives required 30 additional years. According to Stokes, "... although the eye armor that [Fair] was working on might be beneficial in decreasing eye injuries, it so impaired a soldier's peripheral vision and his ability to defend himself otherwise, that it was not practical in battle" (Stokes, personal communication, 1986). Despite the proposal to test various types of commercial safety glasses, no trials were conducted in Korea [50].

The next significant attempt was made in 1962 by McNair, who advocated the development of a polycarbonate eye protective device for the infantryman based on the polycarbonate lenses provided to aviators [51]. This attempt was rejected by US Army commanders, who stated that "the line officers had enough trouble getting the foot soldier even to wear his helmet let alone to have him wear protective glasses or a shield" (McNair, personal communication, 1987). Nonetheless, in 1962, a joint effort by the Quartermaster and the Army Medical Department to develop eye armor was begun. It was to be an optically clear device suitably curved to provide maximum protection with minimum interference with soldiers' activities and include provision for optical correction. A major shortcoming of this effort was the absence of a formally approved Army statement of need for eye armor, and in fact such a "requirement document" was not generated until 1984.

Scientific studies of great relevance were conducted during this period by Stewart and Rose [52, 53] and Williams [54] who, disturbingly, demonstrated that non-heat-treated glass lenses were more protective than heat-treated ones against small missiles and that, under some circumstances, eyes were probably safer uncovered than "protected" by glass lenses. Bryant [55] substantiated the greater impact resistance of plastic (allyl resin) lenses compared to tempered glass lenses. Fackler et al. [56] studied wound ballistics and Davis [57] made valuable observations regarding the optical factors of plano lenses. The major development of the period, however, was the production of optical-grade polycarbonate by General Electric [58]. The marked advantage of polycarbonate over other lens materials was promptly appreciated and it has become the eye and face protective material of choice [59, 60]. Polycarbonate could withstand the impact not only of molten metal but also of a quarter-inch diameter steel ball moving at velocities of up to 500 ft/s [61]. Such lenses of 2.47 mm thickness resisted the impact of 545 mg lead spheres and slugs with pointed heads traveling at 595 ft/s [62].

The Vietnam War Era (1962–1969)

The overwhelming majority of emmetropic infantrymen entered combat in Vietnam without eye protection of any kind. Some drivers of large vehicles and helicopter loaders were provided the US M1944 sun-wind-dust goggle, which, as had been true in World War II, provided only minimal protection from the small fragment threat because its lenses were made of 1 mm thick cellulose acetate. When struck with a fragment, this material readily disintegrated into small sharp-edged fragments (spall) that could themselves damage the eye [63]. The US Army aviator's visor was attached to the standard M1 helmet by Navy researchers in an attempt to protect the eyes of sailors serving on patrol boats in the Delta region of South Vietnam [64]. The visor was judged to be sailor-acceptable, protective, and capable of satisfactorily withstanding the deleterious effects of salt air and intense sun. Although US Army personnel were also equipped with the M1 helmet, no effort was made to evaluate the effectiveness of the helmet-mounted visor for them.

Once again, the ocular threat came predominantly from small, low-velocity missiles. Bryant [65], studying the lens retention of safety frames, concluded, "Polycarbonate plastic lenses exhibited a highly significant increased fracture resistance compared to industrial or dress thicknesses of tempered glass and CR-39 plastic lenses."

Later reviews of wound data and foreign bodies from Vietnam led to the conclusion that the majority of foreign bodies that resulted in eye injury would have been stopped by 2 mm thick eye armor [66–69]. Thus, polycarbonate plastic lenses appeared to have a great potential for truly effective eye protection against flying missiles. Solution of the lens retention and scratch-resistance problems, among others, had to be achieved to permit a complete realization of this potential.

Modern Eye Armor Development: 1969 to Present

The modern development of eye armor began by the testing of the Postoperative Eye Guard (Younger Manufacturing Company, Los Angeles, CA) by La Piana. Intended to protect an eye that had recently undergone cataract extraction, the protective qualities of these devices was demonstrated in demolition tests. Further testing conducted on soldiers during combat training exercises in Vietnam revealed a general dissatisfaction with the plastic ring on the back surface of the Postoperative Eye Guard (the ring was designed to hold the aphakic correction) because of its interference with their peripheral vision. Other frequently expressed complaints were of distortion in the far peripheral field (due to the cylindrical lens power in the lateral portion of the shield) and lack of firm stabilization of the shield on the face when sweating occurred (unpublished data, 1970).

An effort was made in 1971 to interest first the Army and then private industry in the development of eye armor, without success. Part of the problem was political: the failure to interest civilian industry may have been influenced by the widespread antimilitary sentiment at the time. In fact, eye armor in the form now being manufactured (injection-molded polycarbonate in a toric-wrap configuration) could have been manufactured in 1971 (LaMarre, personal communication, 1987). Eye armor development was therefore in a sense another casualty of the Vietnam War, reminding us of Hirschberg's observation that "the history of medicine is part of the history of the entire civilization" [70].

Much effort has been devoted to convincing US Department of Defense (DOD) workers involved in eye armor development that the threat to the eye of the soldier in peace and in war is overwhelmingly from small missiles of 100 mg or less and that eye armor should be developed to protect against this threat. Many DOD workers were unrealistically calling for eye armor that could also protect against larger missiles, including bullets. In Desert Storm, not a single bullet injury was noted in a series of 160 American eye casualties [71]. Such unrealistic demands on the performance of eye armor only delayed the deployment of protection from the much more likely small missile threat.

In the 1970s, studies had demonstrated the superior impact resistance of polycarbonate but also demonstrated degradation in its strength when a scratch-resistant coating was applied [72]. This was a matter of great importance since polycarbonate must be so coated because it is otherwise easily scratched. Further studies demonstrated that polycarbonate lenses could protect the wearer from the small missile threat [66–69]. Among the findings, it was demonstrated that at 30 m from a munitions burst, a polycarbonate eye shield could protect a soldier's eye from most (about 80%) of the fragments.

Prescription polycarbonate lenses became available in 1977 and their advantages over lenses made of glass or CR-39 were noted, including greater impact resistance, higher refractive index (making possible stronger lenses with either less curvature, thinner edges, or both), and low specific gravity (making polycarbonate prescription-bearing lenses approximately one-half the weight of an equivalent strength glass lens) [73]. The increased lateral chromatic aberration of polycarbonate was a relative disadvantage, however, because patients wearing lenses greater than 2 diopters may appreciate colored fringes along black-edged borders [74].

A major conference on Combat Ocular Problems was held in 1980, and much attention was paid to the protection of the soldier's eye from all identified threats [75]. Partially as

a result of this conference, the three following important decisions were made: (1) to link laser eye protection to missile and blunt-force protection, (2) to make polycarbonate the material upon which all development efforts would center, and (3) to provide protection against the missile and blunt-force threat as soon as such became available, and not delay its provision until laser protection became available, as it was judged that the latter required much more time and effort than the former.

The need to protect the soldier's eye from laser wavelengths has concerned the US Army since the advent of this powerful and versatile directed energy source [76]. Many medium-power laser systems are being used in tactical military ground and airborne applications, which include range finding, target designation, ordnance guidance and, during periods of darkness, night vision illuminators. Viewing the collimated laser beam or the specularly reflected beam through a telescope or binoculars can increase the retinal irradiance considerably. Thus, at locations where a laser might be considered safe to view by the unaided eye, it may not be safe when viewed through optical devices. Damage to the eye on the battlefield or the training ground can occur at distances of 400–4000 m depending on the wavelength and power employed, whereas the M-16 rifle (the standard infantry weapon in Vietnam) is effective to only 400 m (Stuck, personal communication, 1986). The inherent ability of polycarbonate to block ultraviolet and far infrared light (such as emitted by the CO_2 laser) added to its attractiveness. The spectral attenuation of a polycarbonate lens in the visible and near-infrared is insignificant, however, and of no value for laser protection in the retinal hazard region (400–1400 nm) [77].

A major stimulus to eye armor development was provided by the appearance on the commercial market of Gargoyles (Pro-tec, Inc., Kent, WA). Gargoyles are fabricated of optical-grade polycarbonate, the thickness of which varies from 2.5 mm in the optical center to 1.8 mm in the periphery and weighs only 1 oz. They are impact-resistant, efficient UV absorbers, and cosmetically acceptable – a very important characteristic because "protective head gear and eyewear will be worn only if the design appeals to the intended wearer" [78, 79]. Gargoyles, or some variation of them, seemed to be an ideal foundation for the development of troop-acceptable eye armor. Some continued to propose the sun-wind-dust goggle fit with 4 mm thick polycarbonate, this despite the fact that the restriction of visual field caused it to be rejected by even many tank crewmen and a similar goggle was rejected by Israeli infantrymen engaged in combat [63].

Testing of Gargoyles on US Army soldiers and Marines began in 1983. Initial results were encouraging, with high troop-acceptance. Several modifications were deemed necessary, however. The nose bridge required strengthening. The distance between the brow and lens had to be increased to minimize fogging. The integrated front had to be extended at least 8 mm posteriorly to provide full protection to the eyes of soldiers with large heads and widely spaced eyes. A polycarbonate lens cleaner was needed because soap and water often are not available in the field.

Because eye armor must protect the ametrope as well as the emmetrope, it was necessary to know the incidence and range of ametropia within the US Army. It had been stated that approximately half of the Army wore glasses [80], but the incidence and degrees of ametropia in different types of units had not been studied adequately. Studies were initiated to determine the incidence and range of ametropia in three Army infantry divisions. The studies substantiated the impression that the incidence of ametropia is lowest in combat arms units, those units whose members are at greatest risk of eye injury in war. This information provided an additional stimulus to work for the development of troop-acceptable eye armor, for it is clear that those most at risk (emmetropic combat arms unit members) had the least, and in most cases no, protection.

The emergence of low-energy lasers as a significant ocular hazard on the modern battlefield gave additional impetus toward the development of eye armor. Whereas up until recently the major threats to the infantryman's eye were ballistic in nature, now electromagnetic energy, in the form of lasers, was a significant and increasing threat.

There have been a number of well-documented laser injuries, usually as a result of incorrect usage of laser range finders, target designators, or other common laser devices utilized by modern armies [81–83]. Added to these accidental exposures are a number of suspected intentional laser exposures over the past two decades, usually directed toward pilots and other aircrew members [81]. There was a documented laser eye injury during the first Gulf War [83]. A number of countries are known or suspected to have developed laser devices with the direct purpose of causing either temporary or permanent eye injury; these countries include the United States, United Kingdom, and the former Soviet Union [81]. Thus, modern eye armor needs to protect against both the ballistic and laser threats.

Contracts were let with the American Optical (Southbridge, MA) and Gentex Corporations (Carbondale, PA) in early 1985, and the American Optical product selected for final development and testing of eye armor. The American Optical eye armor, termed the ballistic and laser-protective spectacle (BLPS), was composed of an integrated front of medium molecular weight polycarbonate containing ultraviolet wavelength inhibitors and coated with an organo-silane for abrasion and chemical resistance. Additional components include a laser-protective device of low-molecular-weight polycarbonate into which are incorporated specific laser wavelength absorbers, a lens carrier, and a retaining strap of neoprene and fabric. Further testing determined that emmetropes preferred a new device manufactured by UVEX (Fürth, Germany), termed SPECS. Unfortunately, SPECS could not be modified to accept a spectacle correction and is unsuitable for use by ametropes. Thus, the US Army began fielding two different forms of eye armor: BLPS for ametropes and SPECS for emmetropes.

In preparation for the invasion of Iraq in 2003, Col William Madigan, the ophthalmology consultant to the Army Surgeon General, recommended that BLPS be provided to US forces prior to deployment [5]. However, this recommendation was not enacted until data of eye injuries suffered by American soldiers were presented in late 2003. Upon review of the data, GEN Schoomaker, the Army Chief of Staff, directed the BLPS to be made a mandatory uniform item for soldiers. Anecdotal evidence provided support for the effectiveness of this mandate, as the rate of eye injuries dropped significantly in populations who consistently wore protective eyewear [84]. After several devastating eye injuries were reported during the battle of Fallujah in 2004, the US Marine Corps leadership also ordered the use of eye armor [84]. Despite high acceptability levels during field testing, ground troops judged the BLPS to be unacceptable. In addition to having an undesirable appearance and feel, the eyewear was also considered to be too complex with too many combinations of lenses and frame types.

The Military Eye Protection System (MEPS) was developed for all ground combat and security personnel at the US Army Soldier Systems Center under a Research and Development program in 2002 [85]. MEPS was a joint Army and Marine Corps initiative with an objective to produce goggles or spectacles with interchangeable lenses with enhanced fit, comfort, and logistical efficiency. The MEPS evolved into the Military Combat Eye Protection (MCEP) program. The MCEP program is tasked with: protecting eyes from external threats (e.g., fragments, electromagnetic radiation, wind, sand, and dust); the provision of corrective lenses for the ametropic service member; encouraging the use of eye projection; and soliciting user feedback in order to respond to evolving threats on the modern battlefield.

The MCEP currently develops and disseminates the Authorized Protective Eyewear List (APEL), a listing of commercial off-the-shelf products that have been approved for military use. To be placed on the APEL, optical products must meet US military ballistic fragmentation standards, which provide five to seven times greater protection for the eyes against ballistic fragmentation than the typical American National Standards Institute (ANSI) Z87.1 standard. In addition, products on the APEL must also meet other MCEP specifications [86] and undergo rigorous field testing, including an evaluation of ease of transport and durability. APEL items are tested as a system including, where appropriate, the polycarbonate prescrip-

tion-bearing lenses. Only those products that meet the standards can receive authorization from the APEL Eyewear Review Board to use the APEL logo. Soldiers are instructed to only use products on the APEL, indicated by a distinctive neon-green APEL logo that is required for all MCEP packaging and the APEL stamp on the left-hand temple or strap [87]. The current APEL, as of March 2017, contains 14 spectacles and 12 goggles [88], reflecting the desire to provide wearers with choices and facilitate usage. A large number of options may also spark increased competition between vendors resulting in improved quality and lower costs.

The lenses must be either clear or provide sun protection and, for some personnel, protective against some laser wavelengths [85]. While emmetropes can use any APEL-approved device, ametropes must use eye protection that can accommodate the Universal Prescription Lens Carrier (UPLC) into which polycarbonate lenses are inserted. Currently, seven spectacle and nine goggle types are suitable for wear by ametropes. In 2014, all MCEP devices began to use a single, universal insert, thus allowing the wearer to be able to switch to a different MCEP model without having to order new lenses. It should be noted that the M50 Joint General Purpose Gas Mask is not on the APEL and its prescription lens insert is not the UPLC [84]. All MCEP with prescription inserts must meet the same performance standards as nonprescription devices.

MCEP is issued to members of the Infantry at Individual Entry Training with their Soldier Protective Individual Equipment as a result of a Rapid Fielding Initiative [85]. The acceptability and overall effectiveness of MCEP among soldiers and operations Iraqi Freedom and Enduring Freedom appears to be higher than previous eye protection development efforts. An MCEP survey conducted in 2009 revealed that 94% of soldiers reported using MCEP on convoys and missions outside the Forward Operating Base [84]. Though not directly tested, recent studies provide initial support for the association between the use of MCEP and reduced ocular injuries (Figs. 16.1 and 16.2) [89–91]. For example, the low proportion of eye injuries (0.5% of all injuries) reported in a study of a mechanized battalion during Iraqi Freedom II was attributed to the almost universal use of eye protection when traveling by vehicle [90]. A 2013 report indicated a sixfold greater incidence of eye injuries in unprotected Afghan soldiers, as compared to accompanying British soldiers wearing APEL eye protection [91].

Fig. 16.2 An example of a large fragment, which certainly would have resulted in significant ocular injury, stopped by modern eye armor

Conclusion

The development of soldier-acceptable eye armor for the American infantryman, seemingly a straightforward, simple task, has in fact required 70 years for successful realization. A thorough understanding of the elements of personal body armor development (missions of the infantryman, threats on the battlefield, materials available for eye protection, mind-sets of both the infantryman and his leaders, and monies for the development, provision, and replacement of eye armor) and the sustained, dedicated efforts of many within and outside the Department of Defense have been required for the development of such eye armor.

In the Iliad, Homer sang, "Men grow tired of sleep, love, singing and dancing sooner than war." As threats to the eye of the soldier (and quite possibly the civilian) evolve, eye armor must also evolve. The development of eye protection for the American infantryman will continue to be a work in progress.

Acknowledgments This work is dedicated to the memory of John Harry King, MD, FACS (COL, MC, USA, Ret), ophthalmologist, soldier, mentor, and friend who serves as an example for all military ophthalmologists.

References

1. Fair J. Protective goggles for the combat soldier. Read before the Meeting of Consultants Ocular Research Unit, Walter Reed Army Medical Center, Washington, D.C.; 1952.
2. Tarassuk L, Blair C, editors. The complete encyclopedia of arms and weapons. New York: Simon and Schuster; 1982. p. 22.
3. Beebe GW, DeBakey ME. Battle casualties. Springfield: Charles Thomas Publisher; 1952. p. 42, 77, 167, 244.
4. Morax V, Moreau F. Etiologie des blessures oculaires par projectiles de querre. Annales D'Oculistique (Paris). 1916;153:321–32.
5. Heier JS, Enzenauer RW, Wintermeyer SF, Delaney M, La Piana FG. Ocular injuries and diseases at a combat support hospital in support of Operations Desert Storm and Desert Shield. Arch Ophthalmol. 1993;111:795–8.
6. Ari AB. Eye injuries of the battlefields of Iraq and Afghanistan: public health implications. Optometry. 2006;77:329–39.
7. La Piana F, Hornblass A. Army ophthalmology in the Vietnam War. The Surgeon General, Department of the Army. Doc Ophthalmol. 1997;93:29–48.
8. Wong TY, Seet MB, Ang CL. Eye injuries in twentieth century warfare: a historical perspective. Surv Ophthalmol. 1997;41:433–59.
9. Vlasov A, Ryan DS, Ludlow S, Weichel ED, Colyer MH. Causes of combat ocular trauma-related blindness from operation Iraqi freedom and enduring freedom. J Trauma Acute Care Surg. 2015;79:S210–5.
10. Neel S. Vietnam studies: medical support of the US Army in Vietnam 1965–70. Washington, D.C.: Department of the Army; 1973. p. 50–51, 55.
11. Aker F, Schroeder DC, Baycar RS. Cause and prevention of maxillofacial war wounds: a historical review. Mil Med. 1983;148:921–7.
12. Tredici TJ. Management of ophthalmic casualties in Southeast Asia. Mil Med. 1968;133:355–62.
13. Evaluation of wound data and munitions effectiveness in Vietnam, vol. 1. Washington, D.C.: US Departments of the Army, Navy and Air Force; 1970. p. D–51.
14. Keeney AH. Lens materials in the prevention of eye injuries. Springfield: Charles C. Thomas Publishers; 1957. p. 62.
15. American national standard practice for occupational and educational eye and face protection, ANZI Z87.1–1 979. New York: American National Standard Institute; 1979.
16. Tarabishy R. Peacetime automatic weapon-related eye injuries: case reports. Mil Med. 1983;148:874–7.
17. McMarlin S, Connelly L. Reforger patient data: information collected in a CSH emergency room during military training exercise. Mil Med. 1985;150:368–71.
18. Dean B. General surgery. In: The medical department of United States Army in the world war (Volume XI: surgery). Washington, D.C.: Government Printing Office; 1927. p. 2, 3.
19. Reister FA. Battle casualties and medical statistics: US Army experience in the Korean War. Washington, D.C.: The Surgeon General, Department of the Army; 1973. p. 48, 51.
20. Coates JB, Randolph ME, Canfield N, editors. Medical Department, United States Army surgery in world war II: ophthalmology and otolaryngology. Washington, D.C.: Office of the Surgeon General, Department of the Army, Government Printing Office; 1957. p. 32, 70, 85.
21. Sliney DH. Standard-item and commercially available laser eye protection, United States Army Environmental Hygiene Agency Nonionizing Radiation Protection Study No. 25–42–0337–86. Aberdeen Proving Ground; 1986.
22. Cotter F, La Piana FG. Eye casualty reduction by eye armor. Mil Med. 1991;156:126–8.
23. Coates JB, Beyer JC, editors. Wound ballistics. Washington, D.C.: Government Printing Office; 1962. p. XVIII, 592–3, 642, 662, 673, 679, 681, 684, 728.
24. Nickel H. Warriors and worthies: armies and armor through the ages. New York: Atheneum; 1969. p. 66–67, 88, 105, 109.
25. Peterson HL. Arms and armor in colonial America 1526–1783. New York: Bramhall House; 1956. p. 5, 106, 111.
26. Dean B. Helmets and body armor in modern warfare including world war II supplement. Tuckahoe, NY: Carl J Pugliese Publisher; 1977. p. 1, 65–66, 145, 186, 234, 236, 237, 287; World War II Supplement, pp 3, 33.
27. Keeney AH. Lens materials and the prevention of eye injuries. Trans Am Ophthalmol Soc. 1956;54:521–65.
28. Held R, editor. Arms and armor annual, vol. 1. Northfield: Digest Books Inc; 1973. p. 306.
29. Dyer G. War. New York: Crown Publishers Inc; 1985. p. 82.
30. Greenwood A, DeSchweinitz GE, Parker WR. Military ophthalmic surgery. Philadelphia/New York: Lea and Febiger; 1918. p. 7, 46, 47.
31. Vail D. Military ophthalmology. Trans Am Acad Ophthalmol Otolaryngol. 1950–1951;55:709–15.
32. Terrien F, Cousin G. Prophylaxie des blessures du globe oculaire. Archives D'Ophtalmologie (Paris). 1914–1915;34:811–7.
33. Whitham LB. Military ophthalmology. Trans Am Ophthalmol Soc. 1919;17:593–716.
34. Link MM, Coleman HA. Medical support of the Army Air Forces in world war II. Washington, D.C.: Government Printing Office; 1955. p. 305, 309, 334.
35. Thomson HC, Mayo L. US Army in world war II the technical services the ordnance department: procurement and supply. Washington, D.C.: Office of the

Chief of Military History, Department of the Army, Government Printing Office; 1960. p. 186.
36. Kuhn HS. Industrial ophthalmology. St Louis: CV Mosby; 1944.
37. Mayer LL. Eyesight in industry. Arch Ophthalmol. 1942;27:375–99.
38. Town AE. Metal eye protector. Arch Ophthalmol. 1943;29:633.
39. Stieren E. A metal safety and glare goggle. JAMA. 1942;120:26.
40. Wurdemann HV. Injuries of the head and eyes in warfare. Mil Surg. 1921;49:443–55.
41. Sweeting CG. Combat flying clothing: Army Air Forces clothing during World war II. Washington, D.C.: Smithsonian Institution Press; 1984.
42. Byrnes VA. Recent advances in military ophthalmology. US Armed Forces Med J. 1951;2:371–81.
43. Sylvia SW, O'Donnell MJ. Uniforms, weapons and equipment of the world war II GI. Orange: Moss Publications; 1982.
44. Cruise R. Protection of the eyes in warfare. Br J Ophthalmol. 1917;1:489–92.
45. Cruise R. Preventable blindness in war. Trans Ophthalmol Soc UK. 1944;64:165–78.
46. Parsons J. Protection of the eyes from war injuries. Trans Ophthalmol Soc UK. 1941;61:157–78.
47. King JH. Research in the Army as it pertains to ophthalmology. Trans Am Acad Ophthalmol Otolaryngol. 1951;55:880–5.
48. Symposium on operative eye surgery and advances in ophthalmology May 18–22, 1953. Washington, D.C.: Army Medical Service Graduate School, Walter Reed Army Medical Center.
49. Fair JR. Eye armor. Am J Ophthalmol. 1957;43: 258–64.
50. King JH. Ophthalmology in the military services. Trans Pa Acad Ophthalmol Otolaryngol. 1955;8:5–10.
51. Lastnik AL, Cleavly BT, Brown JR. Development and fabrication of a polycarbonate eyeshield for the US Army Flyer's Helmet, United States Army Natick Laboratories technical report 71-3-CE. Natick: United States Amy Natick Laboratories; 1970.
52. Rose HW, Stewart GM. Eye protection against small high-speed missiles. Trans Am Acad Ophthalmol Otolaryngol. 1957;61:404–10.
53. Stewart GM. Eye protection against small high-speed missiles. Am J Ophthalmol. 1961;51:80–7.
54. Williams RL, Stewart GM. Ballistic studies in eye protection. Am J Ophthalmol. 1964;53:453–64.
55. Bryant RJ. Ballistic testing of spectacle lenses. Am J Optom Arch Am Acad Optom. 1969;46:84–95.
56. Fackler ML, Bellamy RF, Malinowski JA. Wounding mechanism of projectiles striking at more than 1.5 km/sec. J Trauma. 1986;26:250–4.
57. Davis JK. The optics of plano lenses. Am J Optom Arch Am Acad Optom. 1957;34:540–56.
58. Modern plastics encyclopedia 1986–1987, vol. 63. New York: McGraw-Hill Pub Co, 1986. p. 39–40.
59. Newton AW. Industrial eye protection – an appraisal of some current safety lens materials. J Inst Eng Aust. 1967;39:163–70.
60. Quam GN, Shea J. An investigation of high impact shields for eyes and face. Environ Control Saf Manag. 1971;141(2):24–5.
61. Duke-Elder S, MacFaul PA. System of ophthalmology, vol. 14. St Louis: CV Mosby; 1972. p. 46–7.
62. Goldsmith W. Projectile impact on glass and polymeric ophthalmic lenses and circular plates. Am J Optom Physiol Optic. 1974;51:807–29.
63. Brand J, Reches M, Carroll MM. Eye protection for armor crewmen. Armor. 1985;94:25–7.
64. Hassett RJ, Hanlein SL, Goeller JE. Protective eye shield against small fragments, United States Naval Ordnance Laboratory NOLTR 70-202. White Oak: United States Naval Ordnance Laboratory; 1970.
65. Bryant RJ. Lens retention performance of safety frames. Am J Optom Arch Am Acad Optom. 1969;46:265–9.
66. Reches M. Improved ballistic eye protection. Aberdeen Proving Ground: US Army Materiel Systems Analysis Activity; 1976.
67. Carey ME, Sacco W, Merkler J. An analysis of fatal and non-fatal head wounds incurred during combat in Vietnam by US forces. Acta Chir Scand Suppl. 1982;508:351–6.
68. Robertson DM. Safety glasses as protection against shotgun pellets. Am J Ophthalmol. 1976;81:671–7.
69. Simmons ST, Krohel GB, Hay PB. Prevention of ocular gunshot injuries using polycarbonate lenses. Ophthalmology. 1984;91:977–83.
70. Hirschberg J. The history of ophthalmology, vol. 1, (trans: Blodi FC). Bonn: Wayenborgh; 1982. p XIII.
71. Mader TH, Aragones JV, Chandler AC, et al. Ocular and ocular adnexal injuries treated by Unites States military ophthalmologists during Operations Desert Shield and Desert Storm. Ophthalmology. 1993;100:1462–7.
72. LaMarre DA. Development of criteria and test methods for eye and face protective devices, DHEW (NIOSH) publication no 78-110. Cincinnati: National Institute for Occupational Safety and Health; 1977.
73. Donato JJ, Rengstorff RH. Polycarbonate ophthalmic lenses for eye protection. Rev Opt. 1979;116: 87–8.
74. Davis JK. A polycarbonate ophthalmic-prescription lens series. Am J Optom Physiol Optic. 1978;55:543–52.
75. Proceedings of combat ocular problems conference, October 20–21, 1980. San Francisco: Letterman Army Institute of Research; 1980. p 94.
76. Sliney DH, Yacovissi R. Control of health hazards from airborne lasers. Aviat Space Environ Med. 1975;46:691–6.
77. Sliney DH. Evaluation of laser protective properties of ballistic plastics, United States Army Environmental Hygiene Agency Nonionizing Radiation Protection Study No. 25-42-0343-84. Aberdeen Proving Ground; 1984.

78. Vinger PF. The eye and sports medicine (Chap. 45). In: Duane TD, editor. Clinical ophthalmology, vol. 5. Philadelphia: Harper and Row Publishers Inc; 1985. p. 1–39.
79. Vinger PF. Sports eye injuries: a preventable disease. Ophthalmology. 1981;88:108–13.
80. Rengstorff RH. Problems with optical inserts in military protective masks. Mil Med. 1980;145:334–7.
81. Anderberg B, Wolbarsht ML. Laser weapons: the dawn of a new military age. New York: Plenum Press; 1992. p. 5–6, 76, 93–94, 140–145, 150–166, 176–190.
82. Kearney JJ, Cohen HB, Stuck BE, Rudd FP, Beresky DE, Wertz FD. Laser injury to multiple retinal foci. Lasers Surg Med. 1987;7:499–502.
83. Harris MD, Lincoln AE, Amoroso PJ, Stuck B, Sliney D. Laser eye injuries in military operations. Aviat Space Environ Med. 2003;74:947–52.
84. Mazzoli R, editor. Military Combat Eye Protection (MCEP): historical antecedents and current status. Frontlines of Eye Care Spring; 2016. p. 1–4.
85. Pattison MD. MCEP and APEL – what does it mean? PowerPoint presentation at the worldwide ocular trauma video teleconference, Bethesda; 2015.
86. Pojeta TJ. Military TJ. Military Combat Eye Protection (MCEP) Program Advanced Planning Briefing for Industry (APBI) 2011. Available at http://nrsdec.natick.army.mil/APBI/Eyewear/Army_-_2011_Apr_22_APBI_Military%20Combat%20Eye%20Protection.pdf.
87. U.S. Army Public Health Command. Military Combat Eye Protection (MCEP) and the Authorized Protective Eyewear List (APEL) [fact sheet] 2014. 2014. Retrieved from https://phc.amedd.army.mil/PHC%20Resource%20Library/MCEP_FS_63-013-0314.pdf.
88. Available at https://peosoldier.army.mil/pmseq/eyewear.asp.
89. Thomas R, McManus JG, Johnson A, Mayer P, Wade C, Holcombe JB. Ocular injury reduction for ocular protection use in current combat operations. J Trauma Acute Care Surg. 2009;66(4):S99–S103.
90. Gondusky JS, Reiter MP. Protecting military convoys in Iraq: an examination of battle injuries sustained by a mechanized battalion during operation Iraqi freedom II. Mil Med. 2005;6:546–9.
91. Parker P, Mossadegh S, McCrory C. A comparison of the IED-related eye injury rate in ANSF and ISAF forces at the UK R3 Hospital, Camp Bastion, 2013. J R Army Med Corps 2013, jramc-2013.

Humanitarian Missions

17

Darrel K. Carlton

Introduction

This chapter has been written as a reference for US military ophthalmologists wishing to lead or participate in US military-affiliated ophthalmic surgical missions. Many ophthalmologists choose the specialty with a strong desire to participate in ophthalmic surgical missions abroad. This has been found to be especially true among US Army, Air Force, and Navy ophthalmologists. Due to an overwhelming worldwide epidemic of blinding and largely curable cataract blindness (Fig. 17.1) in the developing world, ophthalmology as a specialty is uniquely positioned to meet this burgeoning humanitarian need, thereby giving US military ophthalmologists a potentially important role to play in meeting this need while providing real-world training.

Doctrinally, the primary purpose of any Medical Readiness Training Exercise (MEDRETE) has always been and still remains the training of US military personnel. It is thus vital for military ophthalmologists wishing to plan and lead an ophthalmic surgical MEDRETE to focus primarily on the unique training value for all mission participants when communicating with other stakeholders during the planning stages. It is hoped that this chapter will encourage US military ophthalmologists to accept the many challenges and many rewards of leading a mission.

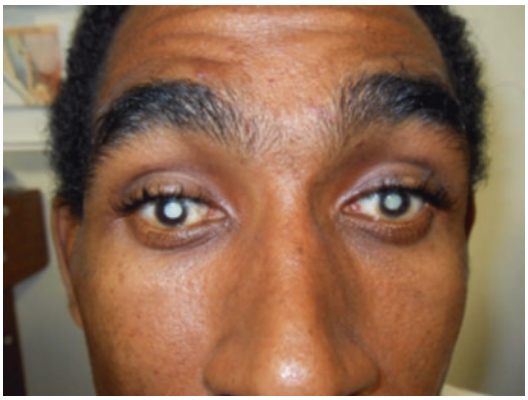

Fig. 17.1 Bilateral blinding cataract in an otherwise healthy young patient; Dominican Republic, 2012

Historical Considerations

Modern US military ophthalmic surgical missions can be traced back to the Vietnam conflict, where Medical Civic Action Program (MEDCAP) exercises were utilized by Army ophthalmologists in treating Vietnamese patients

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and in training their Vietnamese Army ophthalmologist counterparts in cataract and oculoplastic surgery [1].

The 1990s and early 2000s saw a definite rise in the number and frequency of US military ophthalmic MEDRETES, which was primarily due to the advent and many deployments of the US Air Force's Mobile Ophthalmic Surgery Team (MOST), under the direction of Col (Ret) Steve Waller, MD and Col (Ret) Jane Ward, MD. From 1991 to 2003 under the MOST concept, 42 missions were conducted in 11 countries, with over 50,000 patients evaluated and over 5000 surgeries performed. Characteristic MOST missions consisted of 4–12 personnel, which comprised of a variable number of staff and resident ophthalmologists, 1 or 2 optometrists, at least 1 anesthesia provider, several ophthalmic technicians, and at least 1 operating room nurse. The variability in team size was based on mission funding and anticipated surgical volume. MOST mission leadership during this period was varied, with many different ophthalmologists having the opportunity. Noteworthy MOST mission officers in charge (OICs) and participants included the aforementioned Col Steve Waller (Ret), MD; Col Jane Ward (Ret), MD; Col Wendell Bauman (Ret), MD; Col David Holck (Ret), MD; Col Randy Mauffray (Ret), MD; Col Gilbert Dean (Ret), MD; Col Miguel Montalvo (Ret), MD; Col William Flynn (Ret), MD; Lt Col Charles Reilly (Ret); and Lt Col Robert Rice (Ret), MD [2].

Clinically, the MOST focused primarily on cataract surgery (traditional extracapsular cataract extraction or ECCE). Other surgeries included strabismus, glaucoma, and external eyelid procedures and, when good follow-up care was available, corneal transplants. Laser surgeries for glaucoma and diabetes were included on many of these deployments. Expert subject matter exchange between Partner-Nation and US ophthalmologists was also an important component of MOST missions [2].

The new millennium saw a continued presence of ophthalmic surgical MEDRETES being conducted by the Army, Air Force, and Navy. From 1999 to 2008, US Army missions consisted of one mission per year based out of Madigan Army Medical Center, Joint Base Lewis-McChord (JBLM), Washington, and led by Col (Ret) Daryl Ainbinder, MD. Surgically, these missions focused primarily on traditional extracapsular cataract extractions, pterygium surgery, strabismus surgery, and some oculoplastics procedures. Meanwhile, the Air Force still conducted missions, but less frequently than during the heyday of the MOST program in the 1990s, averaging about one mission per year from approximately 2005 to 2012 with a similar case mix to the Army. Finally, the US Navy has conducted numerous humanitarian ophthalmic campaigns aboard both the Hospital Ships *Comfort* and *Mercy*.

The year 2009 saw the introduction of Manual Small Incision Cataract Surgery (MSICS) into US Army missions, under the direction of Col (Ret) Bill Wilson, MD. The improved surgical outcomes of MSICS when compared to traditional ECCE led to a spike in the demand for and the number of Army missions. Dr. Wilson had a vast experience base (over 10,000 cases) in the MSICS technique that he had acquired and mastered during a break in service in Papua New Guinea and in sub-Saharan Africa. This impressive experience base, combined with his skills as a surgical educator, enabled Dr. Wilson to train a cadre of Army ophthalmologists in the MSICS technique. Subsequently, from 2009 to 2016 the Army conducted 18 ophthalmic MEDRETES to 8 countries, all based out of JBLM, in which over 20,000 patient evaluations and over 4000 surgeries were performed.

Army Missions 2009-2017

- 2009: Honduras (214 surgeries), Trinidad (79 surgeries)
- 2010: Honduras (280 surgeries), Dominican Republic (315 surgeries)
- 2011: Honduras (205 surgeries), Malawi (351 surgeries), Dominican Republic (400 surgeries), El Salvador (133 surgeries)
- 2012: Zanzibar (230 surgeries), Honduras (200 surgeries), Dominican Republic (378 surgeries), Burkina Faso (213 surgeries)
- 2013: Mauritania (113 surgeries), two missions cancelled due to sequestration and budget issues
- 2014: Honduras (207 surgeries), one mission cancelled due to personnel constraints
- 2015: Panama (Integrated Army/USAF mission, 254 surgeries), Dominican Republic (207 surgeries, also USAF support)
- 2016: Dominican Republic (221 surgeries)
- 2017: Panama (210 surgeries, USAF led with MEDCOM/USAR support)

The above list highlights missions in which Army ophthalmologists had significant involvement during the 2009–2017 time period. The reader will note the inclusion of numerous missions that did not occur due to various issues, such as budget constraints, sequestration, and personnel issues; issues that highlight the fact that all missions do not occur as originally scheduled. Also noteworthy was the frequent participation of US Army Reserve (USAR) personnel assets during this time frame. In numerous ways USAR Soldier participation in these missions is beneficial, providing an ideal real-world two-week training opportunity (Reservists have a 2-week Annual Training requirement) for the deploying Reservists, overcoming MEDCOM personnel shortfalls, and the exposure to diverse clinical and military experiences that USAR Soldiers bring to any mission.

Leadership of the Army missions during this time frame transitioned from LTC John Thordsen, MD (2009–2010), COL (Ret) Bill Wilson (2011–2012), and COL Darrel Carlton (2012–present). Additionally, over 360 US personnel were trained and deployed, while it is estimated that another 500 personnel that remained in garrison were involved in these deployments. Additional noteworthy ophthalmologist participants in Army missions during this time period include Lt Col Keith Dahlhauser, MD (former USAF); COL Mark Nelson (Ret); COL Anthony Johnson (Ret); LTC Brett Nelson, MD; LTC Patrick Munson, MD; LTC Benjamin Smith, MD; MAJ Bennett Oberg, DO; LTC Erin Seefeldt, MD; and LTC Samantha Rogers, MD.

An exceptional accomplishment in the area of increasing Partner-Nation capacity as a result of the Army's program during the 2012–2013 time frame occurred during a mission to the West African nation of Burkina Faso in 2012, in which Partner-Nation Army ophthalmologist Colonel Jean Diallo, MD, an already outstanding traditional ECCE surgeon, learned the MSICS

Fig. 17.2 Dr. Wilson teaching Dr. Diallo the MSICS technique in Burkina Faso; September 2012

Fig. 17.3 Poster presentation at the AAO annual meeting New Orleans, 2013 with Col Jean Diallo and Col Darrel Carlton highlighting accomplishments and the improved results his patients received

technique under Dr. Wilson's tutelage. Six short months later, after performing roughly 200 MSICS cases on his own, Dr. Diallo was able to not only participate in the next Army mission to West Africa, this time to Mauritania, but he also was that mission's finest MSICS surgeon, and was also able to introduce the technique to numerous Mauritanian ophthalmologists. Figures 17.2 and 17.3 highlight this experience**.**

The importance of the development of the MSICS technique for cataract surgery in the developing world, with visual outcomes that rival phacoemulsification in experienced hands, cannot be overstated. The problem lies in obtaining "experienced" hands in the United States, where modern phacoemulsification machines can remove even the densest lenses and MSICS is rarely, if ever, taught to resident ophthalmologists. Thus, it is only possible to truly become an expert in MSICS, or any surgery, for that matter, if one performs the surgery frequently enough to gain expertise. This, for MSICS means that for military ophthalmologists, deployments on ophthalmic surgical MEDRETES and performing a sufficient number of MSICS cases are the only way to become facile with the procedure.Fortunately, dovetailing with the trauma repair and wartime readiness training foundation that forms the rationale for conducting these

missions, the MSICS procedure itself is outstanding training for ocular trauma care. The fine-tissue manipulation, wound creation, and wound closure skills found in competent MSICS surgeons translates extremely well to repair of ocular trauma. This surely benefits the military ophthalmologists participating in these missions, but also provides excellent training for all mission ancillary personnel, as the ocular surgical care provided by anesthesia providers, nursing personnel, and surgical technicians is nearly identical to combat surgical eye care.

It was with this type of training in mind that Air Force ophthalmologist Lt Col Richard Townley, MD, knowing that he was scheduled to soon deploy to Afghanistan, sought to and participated in an Army mission to Honduras in 2012. A corneal-fellowship trained ophthalmologist, Dr. Townley learned the intricacies of MSICS during this deployment under Keith Dahlhauser, MD (former Lt Col, USAF, MOST veteran, and MSICS expert), and shortly afterward deployed to Afghanistan, where he was able to perform several hundred MSICS cases. After returning stateside, Lt Col Townley took over leadership of the Air Force ophthalmic surgical mission program as an expert MSICS surgeon. From 2012 to 2018, Lt Col Townley led eight AF and participated in two Army missions, the majority of which consisted of mixed AF/Army personnel. While none of these missions were considered "joint," their integrated nature has proven beneficial both in terms of interservice operability and in filling team rosters with qualified volunteers from both services.

Mission Funding

Humanitarian and Civic Assistance, or HCA, programs are provided funding under US Code Title 10, Section 401. These activities promote the specific operational skills of the members of the armed forces and simultaneously create humanitarian benefit to the local populace of the host nation [2]. Note the primary focus on "specific operational skills of the members of the armed forces." HCA funding is generally not provided to exercises deemed to be predominantly humanitarian in nature.

Currently, all MEDRETE mission funding is provided by the Combatant Commands, or COCOMs (i.e., US Southern Command, US Africa Command), through their subordinate Service Specific Commands, or SSCs (i.e., US Army South, US Army Africa, US Air Force South, US Navy South). Prior to 2017, however, this was not the case, at least in the Army, and dual-funding for HCA missions was the norm; with US Army MEDCOM providing HCA funds for shipping of equipment and mission personnel travel and per diem expenses, while the SSC was responsible for HCA funding for expendable mission supplies. The move to full COCOM funding represented positive change, since missions occurring within a specific COCOM should be the full responsibility of that COCOM, with no questions of who is responsible for what during a mission.

Types of Military Ophthalmic Surgical Missions

Currently there are two primary types of missions, small stand-alone (ophthalmic only) missions, and much larger named and typically annual multipurpose missions. As the name suggests, stand-alone missions are ophthalmology-only in nature. Historically, the majority (about 75%) of military ophthalmic surgical missions have been stand-alone missions, but there has been a trend toward increased ophthalmic surgical participation in the larger multipurpose named exercises since 2015. The primary advantages of a stand-alone mission are the greater autonomy afforded to the mission Officer-in-Charge (OIC), a shorter mission planning phase, and that the primary mission focus is on ophthalmic surgeries. The increased level of autonomy can also be a disadvantage if the OIC is inexperienced, or if events on-the-ground require higher-level assistance.

Typical Team Makeup (15–25 PAX)

- Organic assets:
  - 6–10 ophthalmologists (from throughout MEDCOM and USAR, usually includes 2–3 experienced SICS surgeons and 4–6 residents)

  - One anesthesiologist (MEDCOM or USAR)
  - 1–2 CRNAs (MEDCOM/USAR, frequently one CRNA student)
  - 2–4 nurses (MEDCOM)
  - 3–5 enlisted personnel (medics and surgical assistants, from MEDCOM/USAR)
- Nonorganic assets:
  - Medical planner (typically from USARSO, USARAF)
  - Medical Liaison Officer (JTF-B)
  - Recent integrated USAF/USA missions (Panama, Dominican Republic)
  - Logistics specialists (typically from USARSO, USARAF, JTF-B)
  - Volunteers

The above table shows the typical team makeup of a stand-alone Army mission circa 2012–2017. Note that the larger team size is a departure from the smaller teams that were characteristic of the majority of MOST missions. Note also that the team makeup is quite diverse, with participants coming from many different sources, to include active-duty Army and Air Force from numerous locations, US Army Reservists, and volunteers (both US and Partner-Nation). Finally, note the robust presence of trainees, not only ophthalmology residents but also CRNA students.

This team diversity may be seen by some to be a disadvantage as opposed to deploying as an already cohesive team, but it is actually an advantage for numerous reasons. First, not emptying the ophthalmic resources of any one Military Treatment Facility (MTF) for a deployment has been shown to not adversely affect garrison patient access to care requirements. Second, at least for US Army Medical Command participants, similarly diverse teams make up the majority of Theater of Operations MTFs, such as the Combat Support Hospitals deployed in support of Operation Iraqi Freedom (OIF) and Operation Enduring Freedom (OEF-Afghanistan), so a two-week MEDRETE is an abbreviated version of a longer deployment to a Combat Theater, providing excellent real-world training in austere OCONUS environments.

Named missions, such as Beyond the Horizons (BTH) (annual US Southern Command/US Army South mission to different countries in Central/South America), or New Horizons (NH) (annual US Southern Command/US Air Force South mission to different countries in Central/South America), tend to be much larger in scope than stand-alone missions. These exercises typically offer training and humanitarian support involving a spectrum of medical and engineer activities. Medically, in addition to ophthalmic surgical care, optometric care, dental care, primary care, and preventive medicine services may be provided. Other surgical specialties may also be involved, to include ENT, plastics, urology, general surgery, and orthopedics. The budgets for named exercises are exponentially larger than stand-alone exercises (numerous millions of dollars for named exercises versus $200,000–$300,000 for stand-alone missions). The increased scope and budgets of these larger missions result in planning phases being significantly longer and more detailed (several years for named missions versus less than one year for stand-alone missions), and with much larger logistics, security, and command/control footprints.

Ophthalmic MEDRETE Training Value

Short of actually being deployed to a combat zone, deploying OCONUS on an ophthalmic surgical mission provides the best available training to prepare all mission participants for future combat missions. There are numerous compelling reasons that support this assertion. First, all participants plan, train, and deploy to austere locations just as they would to a combat theater. Second, all participants directly work with Partner-Nation personnel and equipment.

Third, staff ophthalmologists and residents gain invaluable experience performing MSICS, ocular suturing, and performing surgery in remote, austere environments. Finally, for the deployed non-ophthalmologist clinical team members, namely, the anesthesia providers, nurses, and eye/scrub technicians, the support they provide ophthalmic surgery on an ophthalmic surgical MEDRETE is nearly identical to what is provided in a Combat Theater MTF, providing an unparalleled training opportunity (Fig. 17.4).

Anatomy of an Ophthalmic Surgical MEDRETE

The best way to gain an understanding of what is required to plan and lead a mission is to break the mission into separate manageable parts. This will be accomplished by first discussing the pre-deployment phase, the execution phase, and finally the post-deployment phase.

Fig. 17.4 Traumatic open globe repair in Malawi by Army resident surgeon (l-r, in purple scrubs Dr. Joseph Msosa, MD, Partner-Nation ophthalmologist; LTC Charles Redger, MD, anesthesiologist; CPT Courtney Crawford, MD, ophthalmology resident; COL Bill Wilson, MD, ophthalmologist); May 2011

Pre-deployment phase For a mission OIC, the pre-deployment phase is by far the most difficult and tedious part of the mission. In order for this first part of the mission to be successful a mission OIC must understand the process by which personnel are tasked to participate in a mission. As alluded to earlier in this chapter, the mission timeline for a large multipurpose named exercise is longer than for a stand-alone mission. The overall requirements, however, are similar; therefore, for the sake of as much simplicity as possible, only one explanation for a "typical" mission timeline will be discussed. Please refer to Fig. 17.5 for a simplified mission tasking sequence.

The first step in any mission to a foreign nation is for that Host (Partner) Nation to formally request support in a given area. This usually occurs well before the conduct of any mission, typically a year or more in advance of the actual deployment. The US Embassy in that nation receives the request and determines how best to address it. Most US Embassies have at least some military presence, most commonly in the form of a Military Group (MILGP). If it is decided that US military assets are best suited to address a request, the Embassy MILGP's Humanitarian Assistance Program (HAP) manager will send the request to whichever COCOM that particular

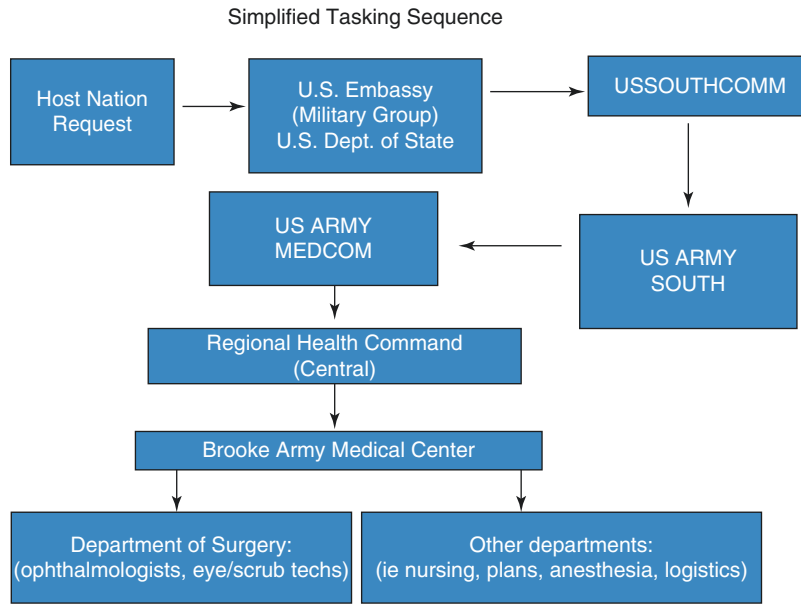

Fig. 17.5 A simplified mission tasking sequence

Partner-Nation falls under (i.e., SOUTHCOMM in the above table), and then the COCOM will task one of their SSCs (i.e., US Army South in the table) to execute the mission. Note that on many occasions US military involvement may not be the best solution to the Partner-Nation request and that other resources, such as the US Agency for International Development (USAID) or Non-Governmental Organization (NGO) support may be the route taken.

Once the decision to conduct a military HCA event is made, the US Embassy team (usually someone from the Military Group) must formally request the personnel support via an OHASIS request. The Overseas Humanitarian Assistance Shared Information System (OHASIS) is a computer system used by the COCOMs and implementing personnel to manage worldwide humanitarian efforts. Since most HAP managers are not well-versed in the types and numbers of, say, ophthalmologists and other clinical personnel required to conduct an ophthalmic surgical MEDRETE, it is up to the prospective ophthalmologist mission OIC to provide the information. For missions to the SOUTHCOMM AOR the Medical Liaison Officers at Joint-Task-Force-Bravo in Honduras have expertise in OHASIS requests and typically assisted the MILGP's HAP managers with this.

An SSC, US Army South, for example, typically has either civilian or military medical planners whose job it is to oversee the planning and execution of HCA operations from the ground up. These individuals typically have extensive knowledge of their SSC's area of operations (AOR) and are an excellent resource when it comes to determining where and when a mission can be conducted. Military ophthalmologists wishing to plan and lead missions must do their best to know these SSC medical planners and maintain frequent enough contact with them to forecast and adequately plan and resource their missions.

From the SSC level via a tasking in one of numerous computerized Training Information Management Systems (i.e., Joint, Army, Navy, Air Force) the tasking for specific medical personnel specialties then will proceed to, for the Army, MEDCOM level, where specific medical operations personnel for a given AOR are present and will aid in directing the tasking to the appropriate medical region within MEDCOM. As with the SSC medical planners, military ophthalmologists involved in the planning and leadership of missions must know and communicate well with these MEDCOM operations officials.

For the larger, named exercises like Beyond the Horizons or New Horizons, the SSC schedules numerous mission planning conferences in the year or so leading up to the exercise itself. For example, the SSC for Beyond the Horizons is US Army South, headquartered at Fort Sam Houston, San Antonio, TX, so the BTH planning conferences are held in San Antonio.

Once it is determined a mission will take place in a given Partner-Nation a Pre-Deployment Site Survey (PDSS) should be conducted to begin mutually deciding upon a myriad of mission details. The initial PDSS should occur as early as possible (minimum of 6 months to a year or more in advance) to give ample mission preparation time for all involved parties. US participants in a typical PDSS may include mission OICs, SSC medical planners, SSC logistics specialists, SSC security specialists, and US Embassy personnel. Partner-Nation participants may include Ministry of Health Officials, military representatives (medical and nonmedical), and hospital representatives for prospective facilities where services may be provided.

For an ophthalmic MEDRETE it is critical that Partner-Nation ophthalmologist points of contact be involved in the PDSS if at all possible, as these individuals will be heavily involved in patient prescreening, determining the scope of the surgical campaign (i.e., cataract/pterygium only, or with strabismus, glaucoma, cornea, or oculoplastics cases), the conduct of the surgical campaign, and patient postoperative care. Past missions in which strong relationships between Partner-Nation ophthalmologists and US military ophthalmologists were not present had a much higher likelihood of encountering issues adversely affecting mission success.

All clinical aspects regarding the prospective care to be provided must be addressed during the PDSS, to include evaluation of operating rooms, storage/security of medical supplies, existing facility personnel (such as number of

ophthalmologists, surgical assistants, anesthesia personnel, hospital leadership and administrative personnel), equipment (such as operating microscopes, vitrectomy machines, autoclaves, microsurgical instruments, and anesthesia machines), interpreter support, and how their facility would handle a needle stick injury to a team member. It is also important to understand how health care in a Partner-Nation is provided. For example, in Panama, Certified Registered Nurse Anesthetists (CRNAs) are not recognized anesthesia providers and therefore should not be taken to missions in Panama. After the PDSS, US Embassy, SSC planners and Partner-Nation officials take the information that was exchanged during the PDSS and work together to create a Memorandum of Agreement (MOA) that outlines in detail various mission responsibilities. While the mission OIC may not be too involved in actual crafting of the mission MOA, he or she must be thoroughly knowledgeable of its contents.

Building a mission team can and should begin early in the pre-deployment phase, perhaps even before the PDSS. Ideally, participation in a mission should be on a voluntary basis and include only trusted personnel with good attitudes and good work ethics. Clinically, the ratio of experienced to inexperienced MSICS surgeons should be 1:1, to assure good patient outcomes while providing excellent training. If there are insufficient expert active-duty MSICS surgeons available, the team leader should inquire about Invitational Travel Orders for a civilian Subject Matter Expert (SME) surgeon. There is a precedent for these requests being granted on numerous prior missions, but they have also been denied. If traveling to a country where other languages are spoken, it is quite helpful to recruit team members fluent in a Partner-Nation language. As discussed earlier in this chapter, building a diverse team from numerous locations is advantageous. All personnel from locations outside the MTF primarily responsible for the mission should gain approval from their chains-of-command well before attempting to generate deployment orders. By name, already-local MTF-approved taskings typically occur much more quickly and easily than "cold-calling" remote regions asking for personnel support, which are not uncommonly denied, and quite frequently delayed.

Logistics issues are, historically, a major threat to ophthalmic surgical MEDRETE success, as expertise in medical and military logistics is usually lacking among military ophthalmologists. It is therefore extremely important to have a mission Non-Commissioned Officer in Charge (NCOIC) who has logistics experience and strong motivation to ensure that all ordered supplies arrive on schedule, are the same supplies that were ordered, and are packed and shipped securely and with all of the necessary Partner-Nation shipping and Customs paperwork. The mission NCOIC is a key team member and must be assigned to the same MTF as the mission OIC. It is also very highly recommended that the ophthalmologist mission OIC visually inspect all mission supplies and equipment prior to and during the load-out along with the NCOIC to assure that all required equipment and ordered supplies are present.

Another pre-deployment logistics issue that can be planned around is to anticipate delays in receiving funding for Class 8 medical supplies, particularly during the first quarter of any fiscal year. This is due to the fact that funds for any fiscal year are usually not disbursed until close to the end of the first quarter (late November–December). It usually takes at least 1 month after ordering supplies to receive them and at least another week or two to inventory and pack them, which means that no missions should be planned until at least the middle of the second quarter of any fiscal year (late January–February). At other times during the fiscal year ordering supplies should be done at least 3 months prior to the deployment.

Timing of the actual shipment of mission supplies varies according to Partner-Nation customs requirements and storage facilities, but mission supplies are typically shipped via government-contracted air commercial carrier around a month prior to 2 weeks prior to the arrival of the Advance Party (ADVON).

The last portion of the pre-deployment phase is devoted to receiving deployment orders and planning travel for all deploying personnel. In order to assure that this process goes smoothly, it is very important that all of those deploy-

ing complete all required Theater and Country-specific training and obtain official passports and visas (if required by the Partner-Nation) well in advance. In fact, all prospective mission participants should always keep current official passports and stay current on all applicable training, even if they are not actively tasked to participate on a mission. It is also important to know your MTF's Plan and Operations section, as the individuals in that section are the ones at your unit level who will handle your tasking and also can assist with numerous pre-deployment requirements, such as ISOPREP, which must be updated within 6 months of any OCONUS deployment.

Execution phase Despite the fact that this phase is characterized by extremely hard work and long hours, this is easily the most enjoyable part of any ophthalmic surgical MEDRETE. It is also the phase during which all of the arduous and tedious work the mission OIC and NCOIC put in during the pre-deployment phase comes to fruition.

The first part of the execution phase begins upon arrival of the Advance Party (ADVON). The ADVON typically consists of the ophthalmologist mission OIC, NCOIC, ophthalmologist Executive Officer (XO), an Operating Room nurse, possibly one other ophthalmologist, and a Medical Liaison Officer from JTF-B (for SOUTHCOMM AOR missions, otherwise a Patient Administration specialist). The ADVON typically arrives on a Tuesday or Wednesday prior to the first Monday of the surgical campaign and the next day begins screening patients that have already been prescreened by the Partner-Nation and scheduling them for surgery. Surgical patient selection is critical, and it is very important to select a strong majority of cataract patients with blinding cataracts (such as the one pictured on the first page of this chapter). It is generally discouraged to offer MSICS to patients with functional correctable vision better than 20/100.

Occurring simultaneously, the NCOIC, OR nurse, and Partner-Nation personnel will be organizing and setting up the operating and storage room(s). Surgical patient screening usually continues until the Saturday when the rest of the deploying team (called the Main Body) arrives.

Once the entire team is present together the ophthalmologist mission OIC gives the team an orientation briefing. If time permits during the Saturday that the Main Body arrives, the entire team is given an orientation of the facility (OR's, storage rooms, etc…) before retiring for the evening. The Sunday prior to the opening of the Surgical Campaign on Monday is reserved for any last-minute OR setup and further team orientation to their workplace for the next 2 weeks.

As mentioned, the surgical campaign is usually 2 weeks in duration, with surgeries scheduled Monday–Friday the first week, Saturday if required by the Partner-Nation, off Sunday, and week two surgeries Monday–Wednesday or Thursday morning. The final Thursday and Friday are devoted to packing supplies and equipment for shipment to CONUS. Saturday the entire team redeploys to their respective home-stations.

Early each postoperative day a team of two to four ophthalmologists and the Medical Liaison Officer are assigned to see the previous day's surgical patients prior to rejoining the rest of the team.

The first day of the surgical campaign is typically challenging and long, as a large team from many different locations and diverse backgrounds learns how to work together and do complex eye surgery in an unfamiliar foreign country using unfamiliar systems under austere conditions. Typically only one to two MSICS cases, and one pterygium case per bed should be scheduled for the first surgical day to account for this steep first-day learning curve, taking the surgical skills of mission surgeons into account. If general anesthesia cases (usually strabismus) are scheduled, only two general anesthesia cases should be scheduled on the first day.

Over the next 2–3 days of the surgical campaign, the number of surgeries scheduled rises to full capacity, given the team's abilities. Each subsequent surgical day brings increases in team cohesion, competence, and confidence (Fig. 17.6).

It is very important not to get overly caught up in number of cases performed, since the primary reason for the deployment is team and individual training, rather than chasing the "record" number of cases, which at some point becomes unattainable and possibly unsafe. Team and patient safety are of paramount importance, and this needs to

Fig. 17.6 l-r CPT Sergev Ujemov, RN, Ft. Carson; Capt Marla Cantrell, RN, Keesler AFB; Maj Maritess Jingco, RN, Eglin AFB; and Maj Petra Holloway, RN, Langley AFB prepare retrobulbar anesthesia syringes before the day's surgeries; Panama, 2018

be frequently stressed to all participating US and Partner-Nation personnel. Additionally, it is important to promote a "see something, say something" mentality amongst team members, whereby any kind of threat to the safety of a patient, Partner-Nation colleague, or team member is brought to someone's attention immediately.

All mission personnel receive role-specific real-world training, with direct supervision from experienced mission ophthalmologists. Since there are several distinct things that occur simultaneously during a cataract surgical MEDRETE, all resident and mission-inexperienced ophthalmologists are assigned different roles each surgical day. This style of training has been very well-received by all trainees.

As mentioned above, all resident ophthalmologists will be part of the postoperative team for at least several days each mission. Determining intraocular lens power via automated keratometry and ultrasonic axial length measurement preoperatively represent another daily assignment. Administration of retrobulbar anesthesia occurs prior to all mission MSICS cases and represents another daily assignment. Finally, all residents are given the opportunity, under direct staff supervision, to perform MSICS to their ability level.

Figures 17.7, 17.8, 17.9, 17.10, 17.11, and 17.12 illustrate some of training roles military ophthalmology residents undertake during a typical mission.

Fig. 17.7 Foreground: San Antonio Military Medical Center ophthalmology resident Capt John Corsini performs automated keratometry measurements on a cataract surgical patient. Background: San Antonio Military Medical Center ophthalmology resident Capt Andrew Pan performs axial length measurements on another cataract surgical patient; Panama, 2018

While the training focus for missions is on US personnel, it is certainly acceptable to exchange

Fig. 17.8 COL Darrel Carlton, MD, San Antonio Military Medical Center, performs a retrobulbar injection while resident-ophthalmologist Capt John Corsini assists and observes; Panama, 2018

Fig. 17.9 Capt John Corsini, MD performs a retrobulbar injection for an MSICS case assisted by Dr. Lourdes Suarez, MD, Panamanian anesthesiologist; Panama, 2018

knowledge with and to train Partner-Nation personnel during ophthalmic surgical MEDRETES. The Partner-Nation capacity-building experience of teaching MSICS to Col Jean Diallo from Burkina Faso was highlighted earlier in this chapter and

Fig. 17.10 l-r Maj Petra Holloway, OR nurse, Langley AFB observes Madigan Army Medical Center (MAMC) ophthalmology CPT Nikhil Godbole, DO perform an MSICS case as MAMC staff ophthalmologist and MSICS expert LTC Erin Seefeldt, MD supervises; Panama, 2018

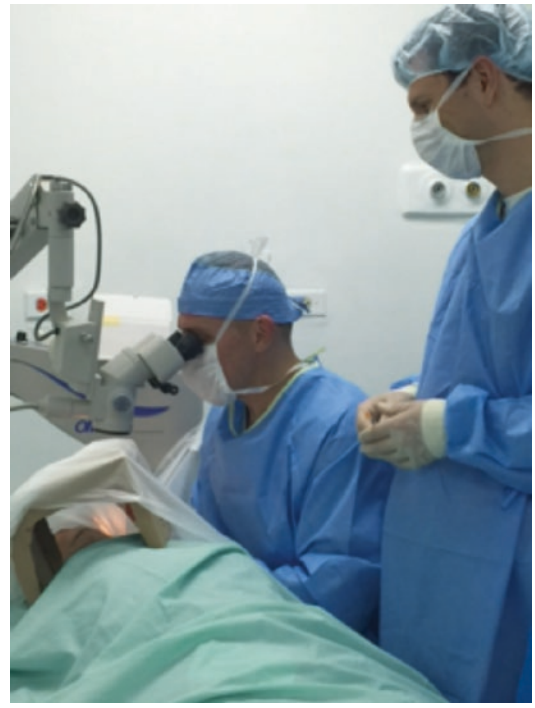

Fig. 17.11 Col Darrel Carlton, MD, SAMMC, performs MSICS while ophthalmology resident Capt (now Maj) James Weightman, MD assists and observes; Panama, 2015

Fig. 17.12 l-r, MAMC ophthalmology resident CPT (now MAJ) Okezi Igboeli, MD performs MSICS while MAMC LTC Patrick Munson assists and supervises; Panama, 2015

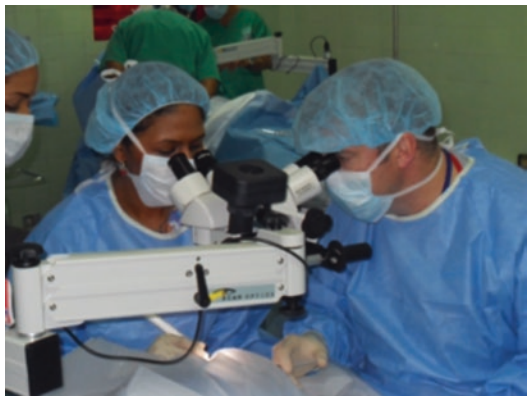

Fig. 17.13 l-r Dominican Republic resident-ophthalmologist Dalma Diaz, MD performs an MSICS case under the direction of COL Mark Nelson, MD, Santo Domingo, Dominican Republic, 2011

was strongly encouraged by US Army Africa officials. Figures 17.13 and 17.14 also illustrate US/Partner-Nation knowledge exchange and capacity building.

Fig. 17.14 l-r Lt Col Richard Townley, MD, San Antonio Military Medical Center teaching Dr. Yvonne Alvarado, MD, Panamanian lead ophthalmologist, Santiago, Panama how to perform a secondary intraocular lens implantation via scleral fixation; Panama, 2015

Complication management during ophthalmic surgical missions is usually straightforward and handled according to standard ophthalmic practice patterns accepted worldwide. Historically, the most common intraoperative complication has been posterior capsular rupture with vitreous loss with a rate of about 3%. In the not-too-distant past the majority of these cases were managed via weck-cell vitrectomy. If weck-cell vitrectomy is still the standard of care for a particular Partner-Nation, this may still be acceptable. However, anterior vitrectomy capabilities are now commonly utilized in the majority of the developing world, making weck-cell vitrectomy obsolete and possessing functional anterior vitrectomy capability standard of care for all US military ophthalmic surgical MEDRETES.

Another fairly complication has been retrobulbar hemorrhages as a result of retrobulbar anesthesia injections, occurring at a rate of about 1%. It has recently been noted that this rate can be lessened significantly if a 2–3-person team approach is used for retrobulbar injections, with one ophthalmologist performing the injection and talking to the patient through the process, another individual providing a fixation target for the patient, and the third person providing interpreter support, if required.

Some controversy exists as to the role of phacoemulsification in US military ophthalmic

MEDRETES. The author of this chapter strongly advises against offering phacoemulsification on these missions for several reasons:

- High cost
- Too high tech, machines easily damaged during transport and by electrical fluctuations
- Partner-Nation indigent patients will not be able to afford phaco after US team departs; Partner-Nation colleagues will most likely only offer phaco to paying patients
- Easier to drop a nucleus than with MSICS, if posterior vitrectomy not available the patient has received a couching procedure with a poor visual prognosis
- MSICS provides outstanding training for trauma-related ophthalmic surgery, phacoemulsification does not

Post-deployment phase The post-deployment phase begins once the team returns to CONUS and only concludes after the mission After Action Report (AAR), any post-mission conferences (typical for named exercises like BTH or NH) are held, mission equipment and supplies have returned, and any postoperative evaluations are complete, and is generally no longer than 2 months in duration.

- AAR – Usually required to the SSC within 30 days after mission is complete. The format is variable, so mission OICs should check with the SSC for proper format. The time to start writing the AAR is during the mission, so everything is still fresh in the AAR-writer's mind.
- Post-mission conferences are typically only held for the large multipurpose exercises like Beyond the Horizons and New Horizons.
- All mission supplies and equipment should be inventoried and, if needed, serviced as soon as possible after returning to CONUS.
- Sending a small component of the team back to the Partner-Nation to conduct postoperative evaluations about 1–2 months after the mission is advisable if at all possible but has only occurred for about 20% of total missions. Historically, this low rate has been due to budgetary issues. There is currently, however, strong support for funding postoperative evaluations at least one COCOM (SOUTHCOMM). The short-term postoperative visual acuity results for US Army ophthalmic surgical MEDRETES have been excellent, but the database would be much more complete if longer-term results were available.

Possible Future Developments

Currently, there is strong support within at least one COCOM (SOUTHCOMM) for increased cooperation with NGOs. There are a multitude of ophthalmic surgical NGOs, such as Surgical Eye Expeditions (SEE), and the Hawaiian Eye Foundation, to name a few, that both teach MSICS and provide ophthalmic surgical outreach to remote nations that have expressed interest in supporting and/or participating in military missions. In fact, an Air Force military ophthalmology resident from SAMCC recently concluded a 2-week mission in the Dominican Republic learning MSICS with the Hawaiian Eye Foundation.

Uniformed Services University (USU) has also been active in the area of developing a "public-private partnership" (PPP) model for a variety of humanitarian services in Guyana and in India by medical professionals in uniform, retired military officers, and US federal employees." A strong PPP component to the development of this model is occurring in Guyana, where corneal specialists Col (Ret) Steven Waller, MD (the same Dr. Waller that developed the MOST program), and Capt (Ret) Joseph Pasternak (US Navy) are working with Guyanese ophthalmologists to develop independent corneal transplantation capability in Guyana [3]. A similar model can easily be visualized utilizing military ophthalmologists as a component of the public–private partnership,

The Defense Health Agency (DHA) already has high-level (O-6) international medicine positions built into their headquarters, suggesting a desire to expand international medical outreach. It

is also hoped that with DHA oversight, increased and improved cooperation between all three services will ultimately lead to truly joint missions.

There currently seems to be more emphasis on Global Ophthalmology in US military residency programs than in the past. The Air Force has already approved a Global Ophthalmology fellowship position and it is hoped that the Army and Navy will soon follow suit.

Conclusion

The worldwide epidemic of curable cataract blindness is not ending anytime soon. It is unlikely that any one nation, any one organization, or any one group of physicians can solve this problem, but it is important for those who can positively impact the situation to do so. This absolutely should include the men and women comprising the population of US military ophthalmology. While a goal in merely participating in a mission is certainly admirable, what are needed most within the ranks are ophthalmologists with the motivation and skill to plan and lead future missions over a long-term basis.

If a reader of this chapter wishes to accept the challenge of mission leadership or just wishes to learn more about mission history or the mission process, he or she can reach out to the author at carltondarr@yahoo.com. If, better yet, said reader has already accepted leadership of a mission and needs very helpful spreadsheets (developed and made available by Lt Col Richard Townley) for both a PDSS and for ordering mission supplies, please email either carltondarr@yahoo.com or rtownley3@gmail.com.

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References

1. Wergeland F. Ophthalmic care of the combat casualty. Textbooks of military medicine. USA: Office of the Surgeon General, Department of the Army; 2003. p. 14–5.
2. Flynn W, Reilly C, Waller S, et al. U.S. Air force mobile ophthalmic surgery team. Mil Med. 2004;169:952–7.
3. Jindal RM, Patel TG, Waller SG. Public-private partnership model to provide humanitarian services in developing countries. J Am Coll Surg. 2017;224(5):988–93.

Appendix: Suturing 101

Introduction

During times of conflict and in the event of natural or man-made disasters, many with unique skills, especially physicians, are called to assist in the treatment of the wounded. In these situations, it is not uncommon for an ophthalmologist whose clinical practice does not involve the traumatic closure of globe injuries to find themselves having to close ruptured globes. Additionally, if resources are especially limited, a surgeon who has never sutured under a microscope, or never closed an ophthalmic wound may find themselves having to do so for the first time.

Unlike other adnexal tissue, the lamellar structure of the cornea imparts a natural stiffness that requires special techniques in order to achieve a watertight, prolate-shaped closure. Below you will find the general principles for suturing globe/cornea injury under a microscope.

Goals

1. Suture equally long and consistent with each suture position, for prolate-shape, peripheral corneal sutures are longer than the central ones.
2. When resuturing a corneal transplant the sutures are placed in a radial fashion – point to the center of the cornea in PKPs.
3. Trauma closure – all sutures placed perpendicular to the laceration.
4. Suture passes, 80–90% depth, and centered on the posterior aspect of the laceration.
5. Suture passes on opposite side of a laceration should be at the same depth, flat and parallel to descemet's.
6. Sutures should achieve good opposition, without excessive compression.
7. For the sake of efficiency, and not compromising the strength of the suture material, try to grab each suture only once for every pass.

Steps

1. Visualize radial line from center of cornea.
   (a) Entry and exit point of needle ~0.7 mm on each side of the interface.
2. Place needle along radial line, or perpendicular to the laceration (Fig. A.1).
3. Place forceps tips along this radial line (Fig. A.2).
   (a) Approach tissue at 45° angle.
   (b) Walk inferior tip down tissue to just above descemet's.
4. Rotate tissue 45° (Figs. A.3, A.4, and A.5).
   (a) Angle needle 45° to the tissue surface.
   (b) Pass needle straight at 45°, first placing tip right behind the forceps.
   (c) Straight line drive, no hand wrist rotation needed (exception: if one of the tissue is significantly swollen) – whole wrist moves horizontally, parallel to floor.

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https://doi.org/10.1007/978-3-030-14437-1

5. Release tissue with forceps (Fig. A.6).
6. Grab and stabilize host tissue, approaching at 45° angle.
7. Gently lower the needle until it is (Fig. A.7)
   (d) Radial to center cornea.
   (e) Flat.
   (f) Parallel to host descemet's.
   (g) If blood in the interface rotate needle up 45°, pull tissues apart, allow your assistant to vigorously irrigate between the tissues, visualize descemet's during irrigation, then rotate needle forward to flat position, lower to level of descemet's.
8. Enter host tissue flat just above descemet's (Fig. A.8)
   (h) As the needle engages tissue, it creates a visual clue "V" at the level of descemet's.
9. Immediately rotate the wrist and needle tip, sharply to abruptly change the needle direction so it exits tissue 90° to the surface `0.7 mm from the laceration (Fig. A.9). If you feel tissue resistance to needle passage, such that you need to provide counterforce with your forceps:
   – Pull the needle back a fraction and turn your wrist more.
   – Needle passing perpendicular to the surface meets little resistance, then you can gently lift needle thru cornea with upward wrist movement (Fig. A.10).
10. Grab the needle below the tip with needle drivers and rotate the needle out of the tissue trying to drag the base of the needle through the precise path as the tip
    (a) Once tissue is cleared bring the needle straight to you (A)
    (b) Until the suture tip is visible at the edge of your surgical field (switch to low power) (B).
    (c) Preposition your typing forceps at position © and grab/stabilize the suture when the tip is visible (C).
    (d) Release the needle while continuing to hold the suture at position C.
    (e) Bring needle driver back into the field over the wound in a wide arching motion (this maneuver prevents inadvertent pulling of the suture through the wound if the needle is caught in the needle driver hinge or if the needle is magnetized and attached to the needle driver.

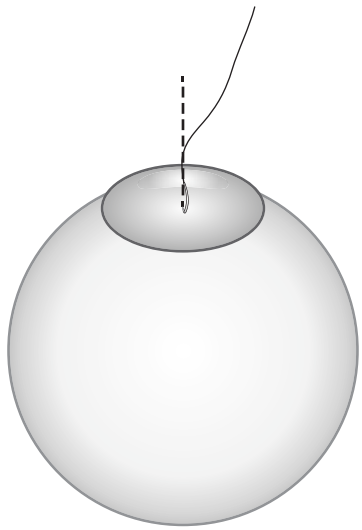
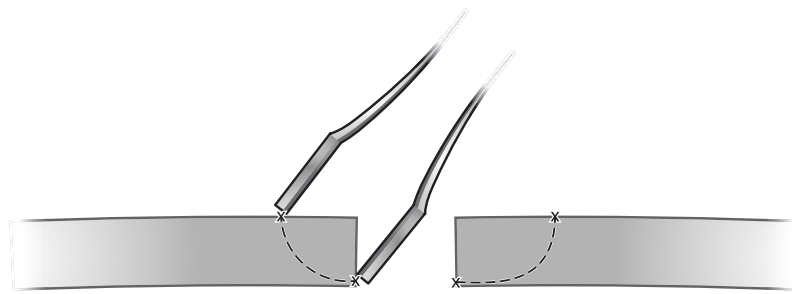


Fig. A.1 Place needle along radial line, or perpendicular to the laceration

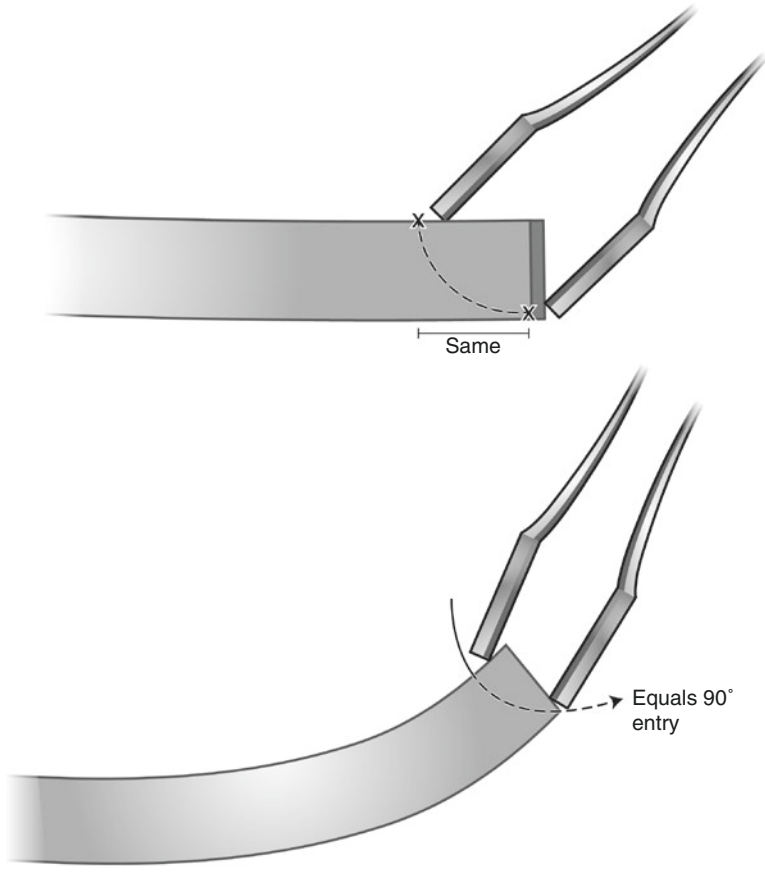
Fig. A.2 Place forceps tips along this radial line



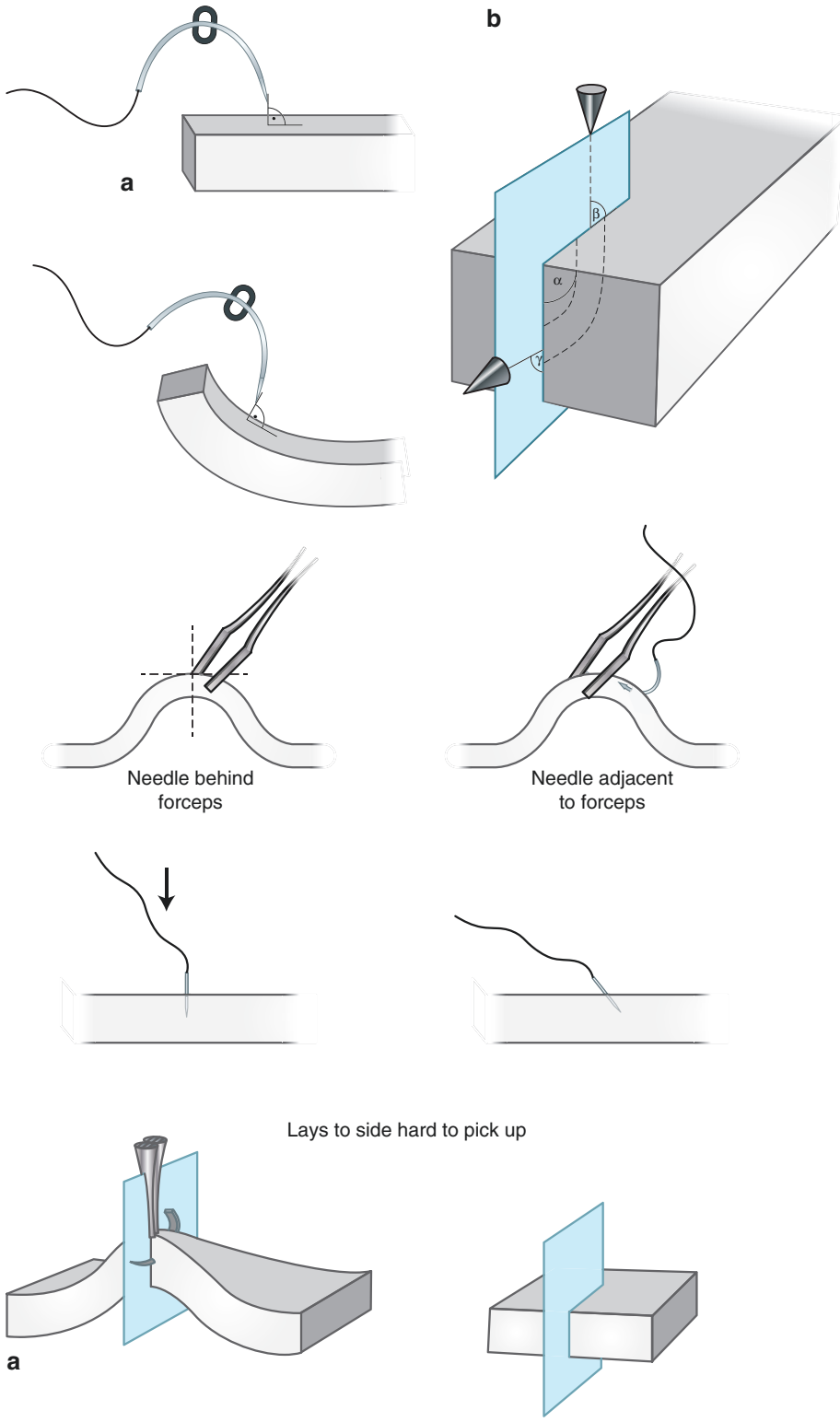
11. Utilizing the forceps at position C pull the suture tip 50% of the distance between the edge of the visible surgical field and the wound. Then raise in preparation for tying.
12. Bring the needle driver back into the field and over the wound (do not release the suture), pointing toward the suture end (Fig. A.11).

3:1:1 or Slip Knot (Fig. A.12)

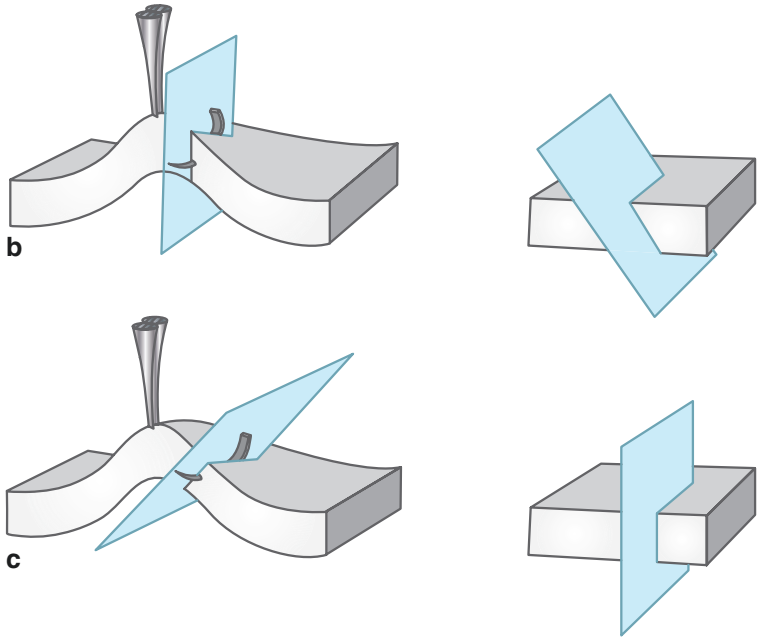
1. When initiating the tying maneuver, the suture, needle driver, and suture tip need to be on the same side of the wound.
   (a) In this configuration the suture tension keeps the suture on the needle driver (Fig. A.13).
   (b) If the needle driver and the suture tip are on opposite sides of the wound, point the needle driver toward the suture tip (if curved needle driver you can twist the needle driver, turning it over). Ask your assistant to put a drop of BSS onto the field to float the suture (Fig. A.14).
2. Wrap the suture around the needle driver with the forceps – this is facilitated if the suture and long axis of the needle driver are parallel.
3. Advance the needle driver, grasp the suture tip (change hands) and lay the suture across the wound with the correct appositional force (Fig. A.15).
4. Now the needle driver and tip are on the same side of the wound again (leave it there).
5. Rotate the needle driver slightly to the side, keeping the tip pointing at the tip of the suture (Fig. A.16) and wrap once with the suture, grab the tip by advancing the needle driver, and pull the suture through the loop.
6. Pull at 90°, equal tension, just above the tissue plane: Do not let the suture tension across the wound change (do not let the looped suture move to either side, the knot should just col-



Figs. A.3, A.4, and A.5 Rotate tissue 45°



Figs. A.3, A.4, and A.5 (continued)



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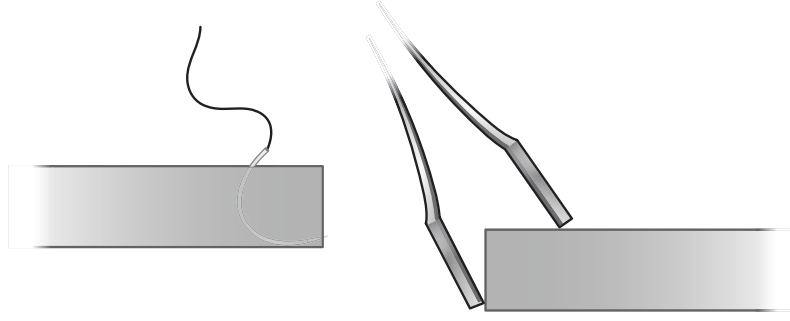


Fig. A.6 Release tissue with forceps

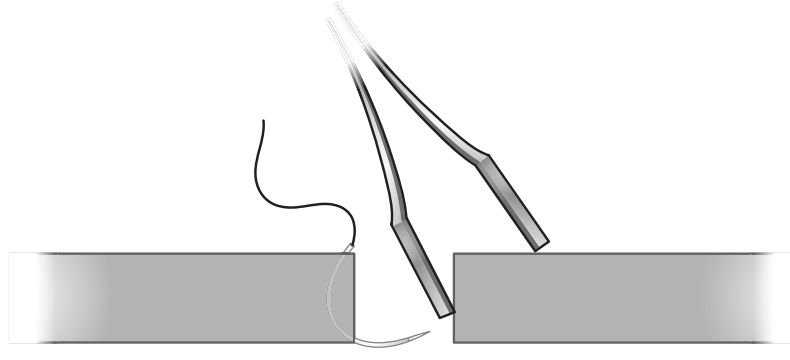


Fig. A.7 Gently lower the needle

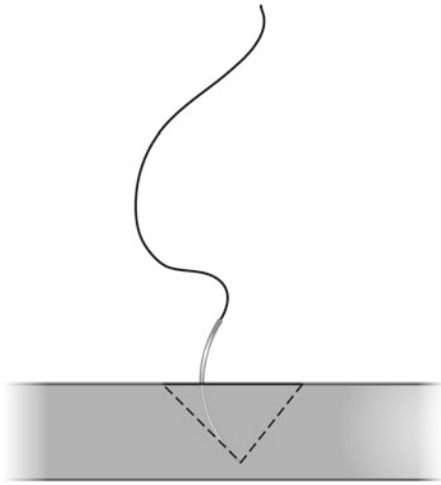


Fig. A.8 Enter host tissue flat just above descemet's

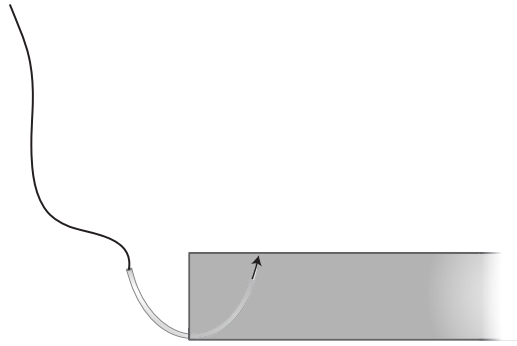
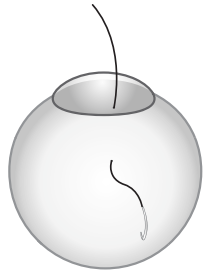


Fig. A.9 Immediately rotate the wrist and needle tip, sharply to abruptly change the needle direction so it exits tissue 90° to the surface `0.7 mm from the laceration

Fig. A.10 Needle passing perpendicular to the surface meets little resistance, then you can gently lift needle through cornea with upward wrist movement



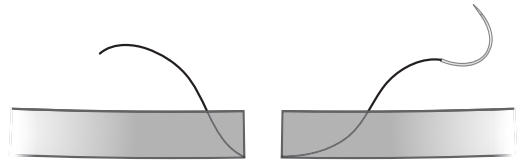
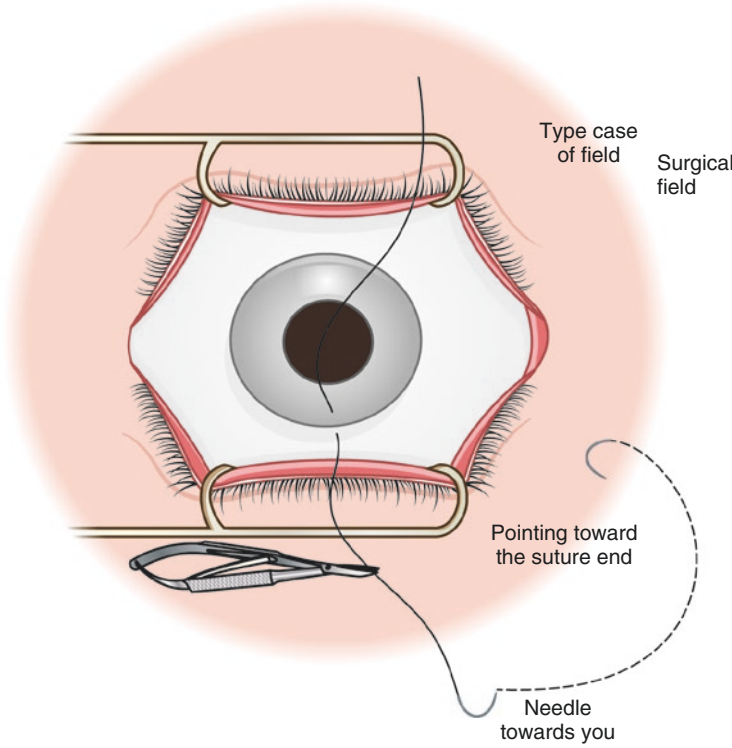


Fig. A.11 Bring the needle driver back into the field and over the wound (do not release the suture), pointing toward the suture end



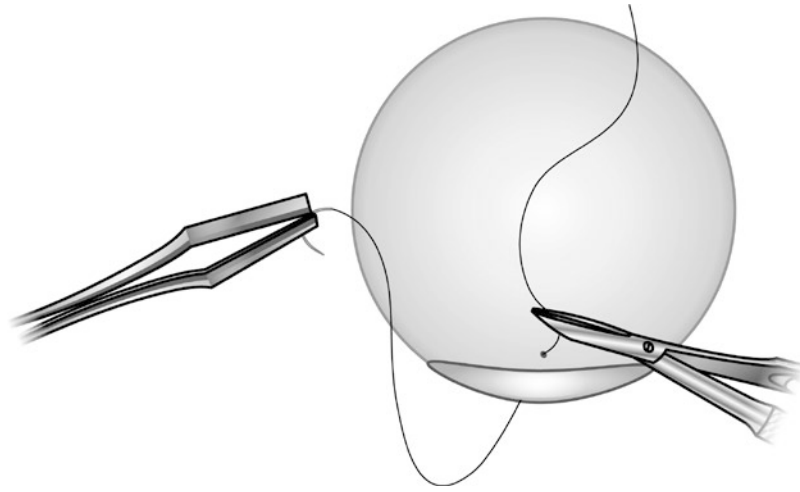


Fig. A.12 Slip knot

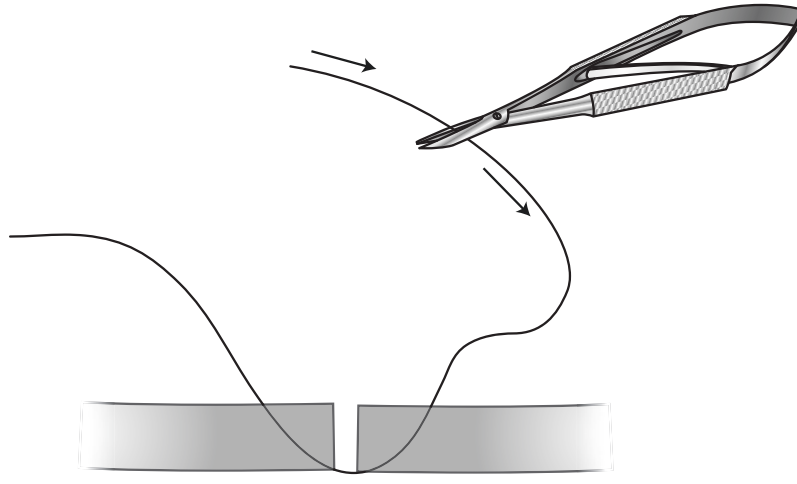


Fig. A.13 In this configuration the suture tension keeps the suture on the needle driver

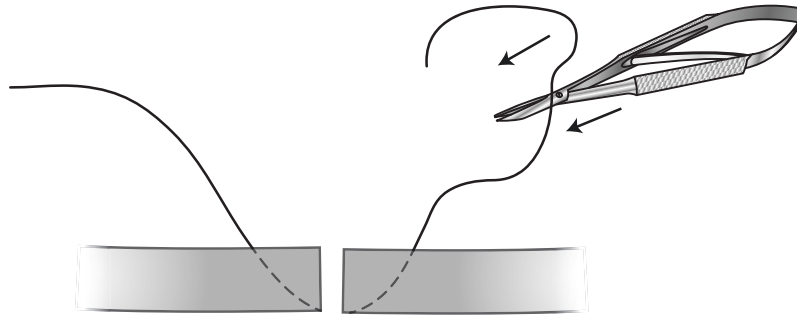


Fig. A.14 If the needle driver and the suture tip are on opposite sides of the wound the suture tension pulls the suture off the needle driver

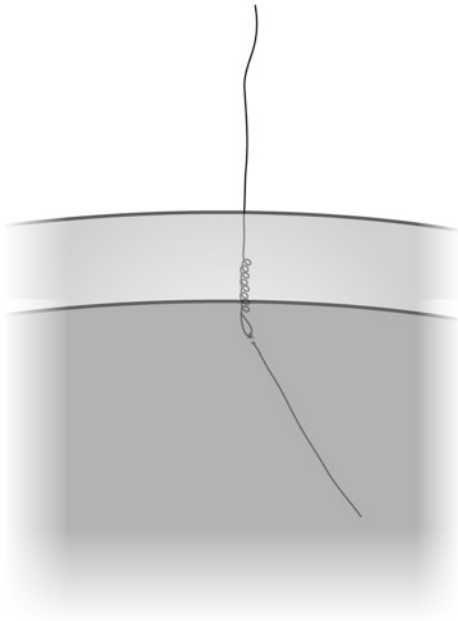


Fig. A.15 Advance the needle driver, grasp the suture tip (change hands) and lay the suture across the wound with the correct appositional force

lapse (Fig. A.17). The tension across the wound is set! One more approximation loop is added by repeating the above steps.

Alternate:

1. Once the correct appositional force is achieved across the wound, keep the suture tension steady in the forceps (At this point you should already have the tip of the suture in your needle driver) (Figs. A.18 and A.19).
2. You can now release the sutures and test the wound for leaking:
   - *If you like the tension:*
     - Lay the short suture end on the cornea.
     - Lift the needle drivers slightly, keeping the tips pointed at the tip of the suture,
     - wrap once, and again tighten at 90° in the plane just above the suture.
   - *If you don't like the tension:*
     - Bring the tip back to the opposite side of the wound, pull equally with both hands and reestablish tension at the desired levels, then repeat the above procedure.

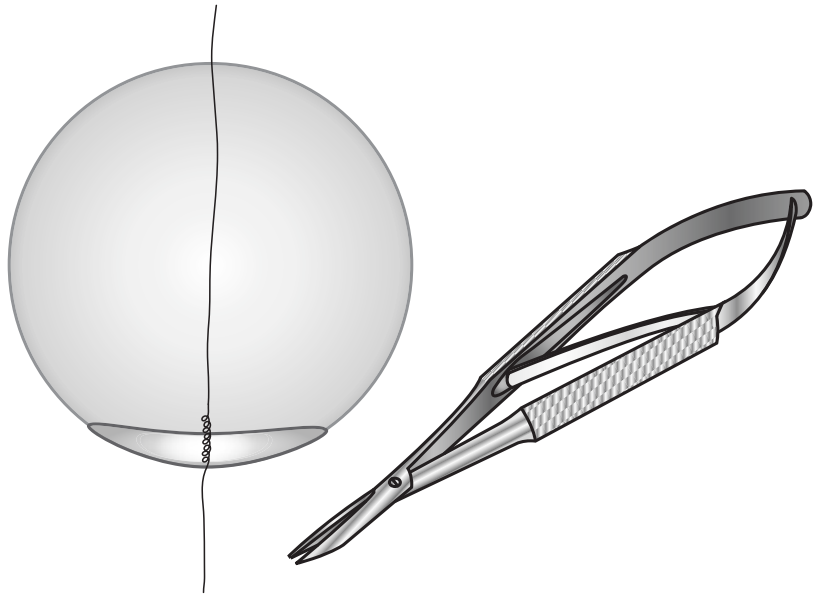


Fig. A.16 Rotate the needle driver slightly to the side, keeping the tip pointing at the tip of the suture

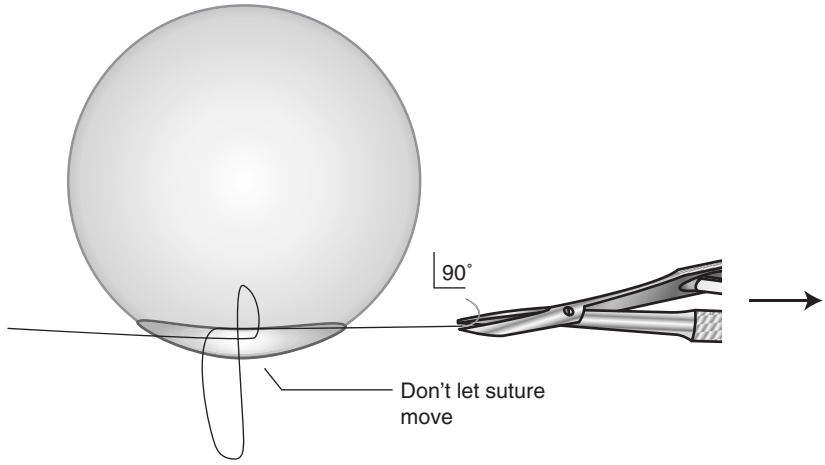
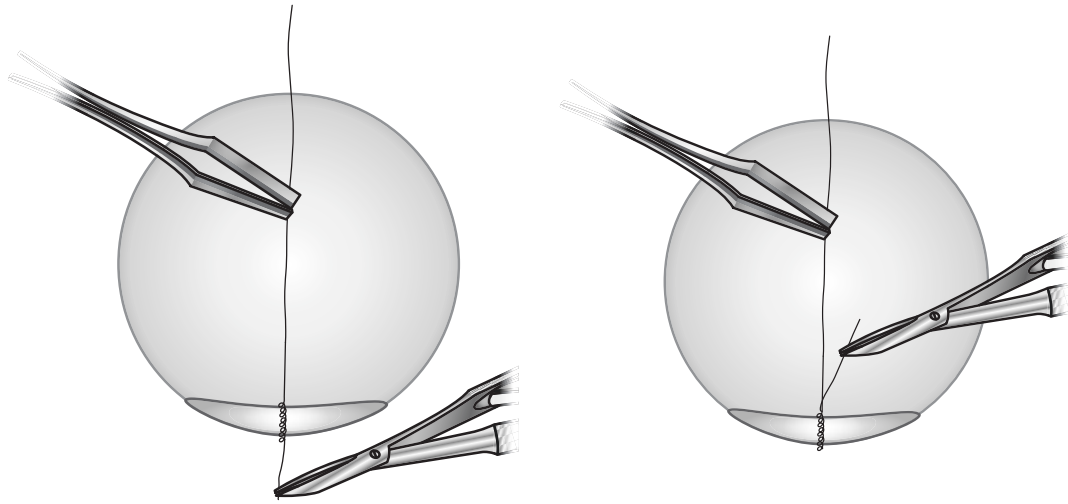


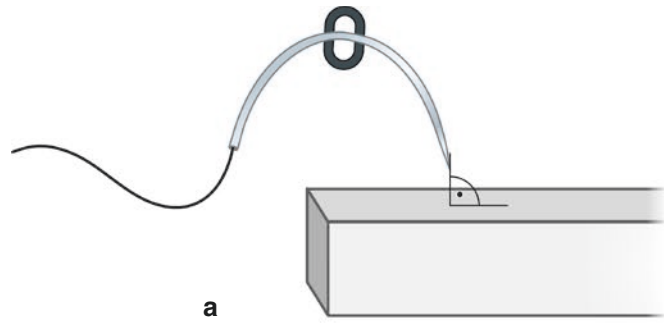
Fig. A.17 Pull at 90°, equal tension, just above the tissue plane: Do not let the suture tension across the wound change (do not let the looped suture move to either side, the knot should just collapse



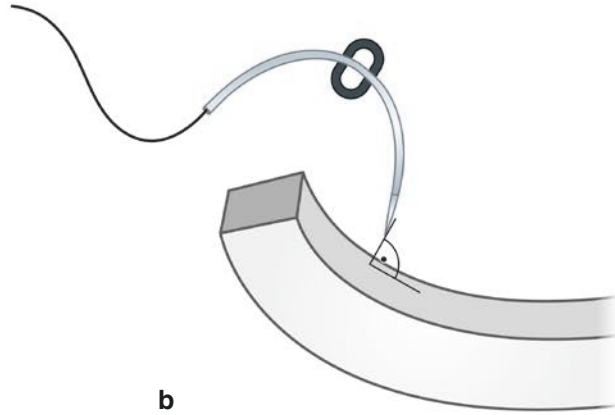
Figs. A.18 and A.19 Without letting go of the suture tip, while keeping tension with the nondominant hand, advance the tip of the suture to the same side of the wound, and create a small knot with a slight jerking motion when the knot has fully collapsed

Backhand Suturing

1. The mechanism of backhand suturing is the same.
2. Passing the needle through the tissue is the same except the needle is away from you.
3. In the initial pass, evert the tissue 45°.
4. The needle approaches the tissue at 45° (no need to turn the wrist) (Fig. A.20).
5. After the needle exits the tissue, bring it toward you just like a standard suture pass, creating an inverted "V" configuration (Fig. A.21).
6. The suture is still stabilized when the tip is at the edge of your view. Then you let go of the needle and make a wide sweep with the needle driver back onto the field.
7. The needle driver is placed across the cornea, and points back into the center. Wrap the suture around the forceps, normally for the initial throw (subsequent directions are unchanged) (Fig. A.22).



a



b

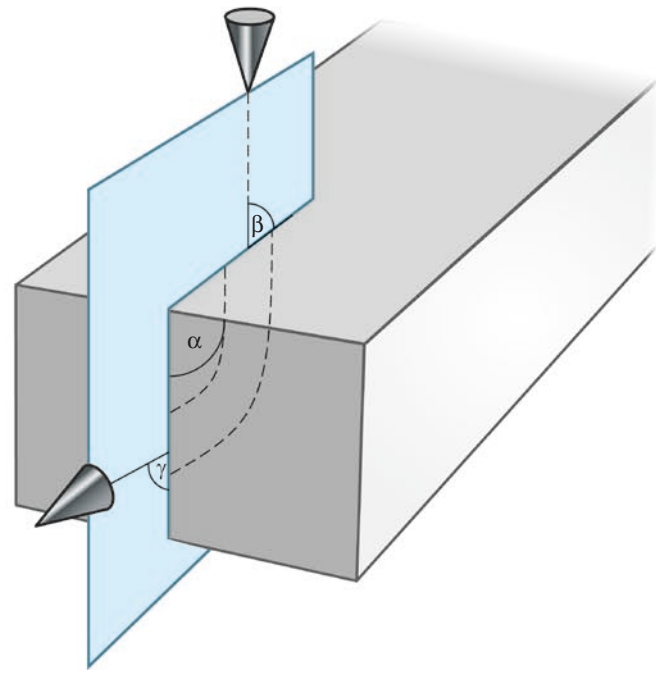


Fig. A.20 All needles should enter the tissue at 90 degrees. This can be accomplished with a no touch technique (**a**), or be everting the tissue at 45 degrees and entering the tissue at 45 degrees to achieve an equivalent 90 degree entry

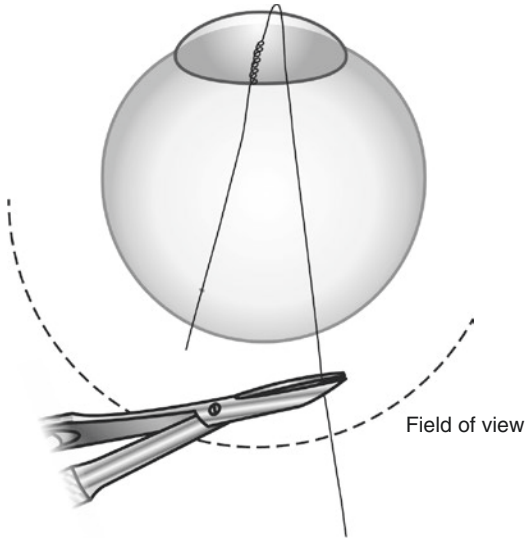


Fig. A.21 After the needle exits the tissue, bring it toward you just like a standard suture pass, creating an inverted "V" configuration

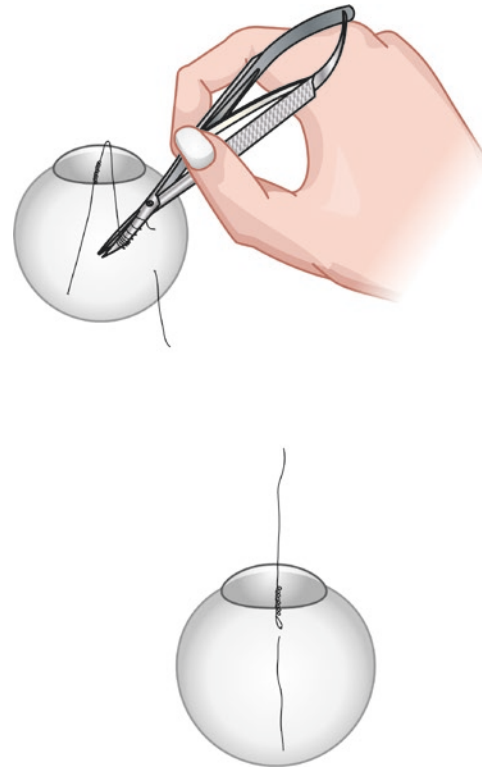


Fig. A.22 The needle driver is placed across the cornea, and points back into the center. Wrap the suture around the forceps normally for the initial throw (subsequent directions are unchanged)

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