Review

The injured eye

Robert Scott*

*Academic Department of Military Surgery and Trauma, Royal Centre for Defence Medicine,
Birmingham, UK*

Eye injuries come at a high cost to society and are avoidable. Ocular blast injuries can be primary, from the blast wave itself; secondary, from fragments carried by the blast wind; tertiary, due to structural collapse or being thrown against a fixed object; or quaternary, from burns and indirect injuries. Ballistic eye protection significantly reduces the incidence of eye injuries and should be encouraged from an early stage in Military training. Management of an injured eye requires meticulous history taking, evaluation of vision that measures the acuity and if there is a relative pupillary defect as well as careful inspection of the eyes, under anaesthetic if necessary. A lateral canthotomy with cantholysis should be performed immediately if there is a sight-threatening retrobulbar haemorrhage. Systemic antibiotics should be prescribed if there is a suspected penetrating or perforating injury. A ruptured globe should be protected by an eye shield. Primary repair of ruptured globes should be performed in a timely fashion. Secondary procedures will often be required at a later date to achieve sight preservation. A poor initial visual acuity is not a guarantee of a poor final result. The final result can be predicted after approximately 3–4 weeks. Future research in eye injuries attempts to reduce scarring and neuronal damage as well as to promote photoreceptor rescue, using post-transcriptional inhibition of cell death pathways and vaccination to promote neural recovery. Where the sight has been lost sensory substitution of a picture from a spectacle mounted video camera to the touch receptors of the tongue can be used to achieve appreciation of the outside world.

Keywords: ballistic; eye; injury

1. INTRODUCTION

The eyes occupy 0.1 per cent of the total and 0.27 per cent of the anterior body surface; the significance of their injury is magnified as vision is the most important sense. Loss of vision is likely to lead to loss of career, major lifestyle changes and disfigurement. Unlike in peacetime where unilateral injuries are the rule, ocular war injuries are bilateral in 15–25% of cases [1,2]. Eye injuries come at a high cost to society and are largely avoidable [3].

Using body armour over the trunk region makes explosions more survivable but leave the face and eyes relatively exposed. Before the twentieth century, the eye casualty rate was less than four times its expected percentage based on body surface area alone. This rate has increased to over 50 times the expected percentage in the late twentieth and early twenty-first centuries. Injured personnel who once would have died of thoracic and abdominal wounds now present with non-fatal injuries to the eyes and extremities (figure 1).

Ocular injuries from terrorist bombings have a similar incidence to those of modern warfare, ranging from 3 per cent in the Manchester bomb to 10 per cent in the Oklahoma City and Dharan blasts. The pattern of injury in these incidents is from missile fragments and small fragments of glass, cement, mortar and

other debris associated with the blast that cause minimal damage if they impact on the clothes or skin, but significant morbidity if they hit the eye [4–6].

There has been an increase in the proportion of war-related eye injuries from less than 2 per cent up to the Second World War; between 2 and 3 per cent up to the Korean War; and between 5 and 7 per cent in the three Arab–Israeli conflicts, attributed to urban and tank warfare [7,8]. A combination of further urbanization of warfare and increased weapon explosive power with relatively poor ocular protection in relation to the rest of the body saw a further increase to 13 per cent in Operation Desert Storm in 1991 [9]. Prioritization of combat eye protection may have helped reduce the proportion of eye injuries to 6 per cent in Operations Iraqi Freedom and Enduring Freedom (2001–2005) [10].

2. TYPES OF BLAST INJURY

(a) *Primary blast injury*

An ocular primary blast injury (PBI) is from an explosive shockwave causing pressure differentials across the different tissues with shearing and, at a particular limit, pathological damage [11–13]. Reflection of the shock wave by the orbit may amplify the effect [14,15]. Other factors that influence the blast effect include the peak overpressure achieved, its duration, the distance from the explosion and if the eyes point towards the explosion.

* Author for correspondence.

One contribution of 20 to a Theme Issue 'Military medicine in the 21st century: pushing the boundaries of combat casualty care'.

Usually, ocular blast injuries are from a combination of primary and secondary processes. Cases of PBI alone occur in those with intraocular lesions without penetrating or perforating injury. That group of patients has remained roughly constant in proportion since the Second World War, at an average of 19.3 per cent of all eye injuries (table 1). The visual prognosis from PBI is poor with a final visual acuity of 6/12 or better in 16–32% of cases, with injuries to the optic nerve, choroid and retina carrying a worse

Figure 1. Traumatic hyphaema with inferior level of blood in anterior chamber.

prognosis than those to the anterior segment, adnexae or intraocular haemorrhages [16–18].

The spectrum of injuries from primary blast is identical to those from blunt contusion injuries and varies with the type of explosive and surrounding conditions. In the 21 cases of PBI reported in the literature the posterior segment is predominantly affected in approximately 57 per cent of cases, even though the blast wave traverses the anterior segment to reach it [16,19]. Conjunctival lacerations and subconjunctival haemorrhages are particularly common [20]. Blast injuries to the ciliary body predispose to ocular hypotony that will usually recover and reduced refractive accommodation that makes unaided reading difficult with associated eyestrain. The lens is often affected by PBI, causing cataracts, which often resolve [21].

In the posterior segment, PBI is associated with vitreous haemorrhage and, more rarely, retinal tears and detachments [22]. Macular pigmentation from retinal pigment epithelial disturbance and retinal swelling is usually asymptomatic, but occasionally atrophic changes occur with profound loss of central vision [23,24].

Berlin's oedema occurs when locally produced metabolites stimulate vasodilatation leading to retinal swelling and haemorrhage. This resorbs over a few days leaving a variable pattern of retinal atrophy. Vision is markedly reduced during the oedematous

Figure 2. Birmingham eye trauma terminology system (BETTS).

Table 1. Incidence of war-related eye injuries from the 19th to 21st century.

war	year	population	% non-fatal injuries
Crimean	1854–1856	British	0.65
Crimean	1854–1856	French	1.75
American Civil	1861–1865	American	0.5
Franco-Prussian	1870–1871	German	0.86
Franco-Prussian	1870–1871	French	0.81
Russo-Turkish	1877–1878	Russian	2.5
Sino-Japanese	1894	unknown	1.2
Russo-Japanese	1904–1905	Japanese	2.22
Russo-Japanese	1904–1905	Russian	2
World War 1	1914–1918	US Army and Navy	2
World War 2	1939–1945	US Army	2
Korean	1950–1953	US Army	2.8
Six-Day	1967	Israel Defence Forces	5.6
October 1973	1973	Israel Defence Forces	6.7
Lebanon 1982	1982	Israel Defence Forces	6.8
Arabian Gulf	1991	US Army	13
Arabian Gulf	2001–2005	US Army	6

Figure 3. Traumatic lens dislocation.

Figure 5. Corneal perforation covered by bandage contact lens.

Figure 4. Choroidal rupture extending to the macula, with secondary epiretinal membrane formation (arrows) and retinal traction (arrowheads).

phase though recovery of some or all the visual function is the rule after a few weeks [25].

Traumatic optic neuropathy is a well recorded, if uncommon, PBI associated with large blast forces [26]. It may follow massive retinal atrophy with axonal loss following widespread retinal oedema, vasoconstriction of the optic nerve blood supply, mechanical disruption of optic nerve axons, or compression from a retrobulbar haemorrhage [27].

(b) *Secondary blast injury*

Secondary blast injuries (SBI) are due to the impact of shrapnel from the explosive device itself or from exogenous debris propelled by the explosion. They are the most common form of high explosive ocular injuries. The projectiles cause penetrating and perforating injuries to the globe and ocular adnexae. Until recently, it was rare to find a case of PBI without any evidence of contusion or any secondary injury to the eye or adnexae [26,28].

The *Birmingham eye trauma terminology system* (BETTS) is a standardized system that should be used to describe and share eye injury information. It is particularly useful with battlefield trauma cases where the casualty is treated by multiple care teams (figure 2) [29].

Figure 6. Penetrating injury to eye from screw.

(c) *Closed globe injuries*

Closed globe injuries are common to both primary and secondary blast injuries. Secondary blast injuries are caused by an impacting force that strikes the eye causing shortening anteroposteriorly and elongation equatorially. The lens–iris diaphragm is displaced posteriorly centrally while the peripheral structures are expanding outwards. This causes tearing of the ocular tissues, particularly in the iridocorneal angle. When the compressing object is larger than the orbit it pushes the globe posteriorly to suddenly increase the orbital pressure. The pressure is relieved by a blow-out fracture of the orbit, typically inferiorly into the maxillary sinus. This often protects the globe from injury though there is up to a 30 per cent incidence of ruptured globe reported in conjunction with orbital fracture [30].

Corneal abrasions are diagnosed with fluorescein staining and a blue light after an anaesthetic drop is instilled to allow examination of the eye. Treatment is with topical antibiotic ointment to lubricate the eye surface and reduce the chance of a secondary infection. The underlying corneal stroma is clear in corneal abrasion; a stromal infiltrate may indicate bacterial or fungal keratitis [31]. Occlusive eye patches are not required [32].

Corneal foreign bodies (FBs) can be intensely painful, if superficial. Radiographic investigation is mandatory to exclude an intraocular FB. Diagnosis is with fluorescein staining after an anaesthetic drop is instilled to allow examination of the eye. There is often a sterile infiltrate surrounding the FB, especially if it has been present for more than 24 h. The eyelids should be everted and if necessary double everted to exclude the presence of multiple FBs. After blast injuries, particularly improvised explosive device (IED) and mine blast injuries there are often multiple deep stromal FBs. After an initial period of inflammation, they characteristically become quiescent and the FBs will remain inert in the corneal stroma with little visual effect. Caspar's rings of the cornea are 0.5–1.1 mm disciform lesions that lie under corneal FBs and resolve within 4–5 days with no harmful effects [33].

Iris and ciliary body contusion injuries may cause traumatic iritis requiring a topical cycloplegic agent to relieve pain. Iridocorneal angle recession is common after blunt trauma and is associated with damage to the trabecular meshwork. It can impede aqueous flow from the eye to cause a rise in the intraocular pressure with secondary glaucoma in 7–9% of cases [34,35]. Patients with angle recession without glaucoma need to be followed annually for the development of glaucoma; patient education is important as the risk is lifelong [36]. Iridodialysis is a detachment of the iris root from the ciliary body and can produce corectopia (displaced pupil) and pseudopolycoria (more than one pupil). Treatment is with direct surgical closure of the defect [37].

Cyclodialysis clefts arise from traumatic separation of the ciliary body from the sclera; this allows aqueous to exit directly to the subchoroidal space and causes ocular hypotony. Diagnosis is by gonioscopy or an anterior segment ultrasound scan. Management is with topical atropine for 6–8 weeks as spontaneous resolution is usual. If it is persistent, transcleral diode laser to the cleft is applied. Direct surgical closure is required if medical treatments fail [38].

Hyphaema describes layering of red blood cells (RBC) in the inferior anterior chamber from a bleed (figure 1). The height from the inferior limbus is measured to guide management and chart progress. Microhyphaema is a small hyphaema in which there are only suspended RBCs in the anterior chamber with no layered clot. The main complications are raised intraocular pressure and rebleeds. Ophthalmic evaluation is required to exclude associated retinal tears (10%), or secondary glaucoma (5%). Corneal blood-staining may occur if there is prolonged hyphaema with a high intraocular pressure. Management is by bed rest with 30° head elevation for 4 days to prevent rebleeds. Sick cell trait and anaemia exacerbates the effect of a hyphaema and should be

excluded, if appropriate. Elective anticoagulants are discontinued and patients put on light activity for two weeks. Topical mydriatics and steroids are prescribed for two weeks. A raised intraocular pressure occurs in 30 per cent of cases and is treated with oral or topical ocular antihypertensives. Diamox is contraindicated if there is sickle cell trait or disease. Rebleeding occurs in 4–40% of cases, usually 2 to 5 days post-injury. Surgical wash-out of the anterior chamber is required for 5 per cent of cases. Iridocorneal angle recession or iridodialysis occurs in 75 per cent of cases, but only 5 per cent go on to develop secondary glaucoma [39].

Lens contusion cataracts are classic of blunt trauma and may not appear for years after the injury, causing glare and loss of vision. Lens subluxation is a partially dislocated crystalline lens caused by incomplete zonular dehiscence from trauma (figure 3). If subluxation occurs after relatively minor trauma, associated Marfan's disease, homocystinuria, Weill–Marchesani syndrome, Ehlers–Danlos syndrome and syphilis should be excluded. In anterior chamber dislocation, the lens should be repositioned after pupil dilation and supine positioning with corneal indentation with a gonioscope at a slit lamp. If there is an intact lens capsule, posterior segment dislocation can be managed conservatively. Surgical removal of traumatic cataracts is by phacoemulsification and lens implantation is performed if there is a visually significant cataract. Traumatic cataracts can be complex cases due to lens zonular dehiscence.

Posterior segment contusion injuries are common after explosions. Commotio retinae (Berlin's oedema) describes the transient grey-white opacification at the level of the deep sensory retina after blunt trauma. It settles over 4–6 weeks leaving retinal pigment epithelial disturbance, retinal atrophy, optic atrophy and on occasion a traumatic macular hole. Visual loss may be transient or permanent according to the extent of macular involvement.

Choroidal ruptures are typically crescent-shaped and sited at the posterior pole (figure 4). They occur when the eye is deformed by a blunt injury. Bruch's membrane of the choroid is relatively non-elastic and ruptures while the more elastic retina and sclera remain intact. They are associated with subretinal haemorrhages that clear to reveal the rupture. Visual recovery is the rule, but can be reduced if the rupture involves the fovea [40].

Sclopetaria, traumatic chorioretinal rupture, is from a concussion injury where an object strikes the eye at high velocity but does not penetrate the eye. The choroid and retina are locally disrupted by the transmitted energy (coup) and at the opposite side of the eye (contrecoup) to give an acute traumatic degeneration of the choroid and retina. There are often dramatic retinal tears with associated shallow retinal detachment. These characteristically do not require active treatment as scarring from the surrounding tissue seals the retinal break.

Optic nerve avulsion and traumatic optic neuropathy can occur after blast injuries. Diagnosis is confirmed by ultrasound or computerized tomography (CT) scan. It is typically associated with a relative afferent

Table 2. Stages of recovery from primary and secondary ocular blast injuries.

stage of recovery post-injury	time	phase of recovery	prognosis
1	0–3 weeks	rapid recovery	poor visual acuity does not preclude good recovery
2	4–12 weeks	specific syndromes	final visual acuity can be predicted at this time
3	3 months–3 years	late changes (rare)	unpredictable but late recovery more than exacerbation

pupillary defect. There is no evidence of superiority of medical (steroid) and surgical (orbital decompression) treatment over observation (see table 2).

Traumatic retinal tears and detachments are managed surgically with retinopexy for retinal tears and vitrectomy and internal tamponade or cryopexy and buckling procedures for retinal detachment. If an intraocular gas is used, the patient cannot travel by air until it has resorbed, in case of sudden gas expansion at altitude. Retinal detachments associated with penetrating trauma is particularly susceptible to post-operative retinal scarring (proliferative vitreoretinopathy). This reduces the anatomical and visual outcomes of surgery and the patient should be warned of the guarded prognosis [41,42].

Retinal dialyses describe a peripheral retinal disinsertion between the edge of the retina and the ora serrata from sudden expansion of the ocular equator from blunt injury. They are responsible for up to 84 per cent of trauma-related retinal detachments. The detachment may evolve slowly, often years after the trauma. Diagnosis is often when central vision is affected from macular involvement or as an incidental finding during routine ophthalmic examination. Traumatic macular holes form as a result of acute changes in vitreoretinal traction over the macular area from ocular contusion; they cause loss of central vision and distortion, but there is a good visual prognosis with successful surgery.

Corneoscleral non-penetrating lamellar lacerations are diagnosed by careful ophthalmoscopy. Siedel's sign indicates a penetrating injury of the anterior segment. Here, a drop of sodium fluorescein dye is applied to the cornea and is observed with a slit lamp's cobalt blue light. Any fluid leaking out of the eye dilutes the yellow fluorescein and can be observed as a lighter green fluorescent wave. Corneal lacerations can be managed with a bandage contact lens if the laceration is small and self-sealing (figure 5). Topical antibiotics must be given for 1–2 weeks after the laceration.

(d) *Open globe injury*

Penetrating eye injuries are sharp eye injuries that have a single entrance wound from the injury (figure 6). If more than one wound is present, each must have been caused by a different agent. Perforating eye injuries have an entrance and exit wound; both wounds are caused by the same agent.

The signs are often obvious with an entry/exit site and extrusion of ocular tissue. If there has been an occult penetration there may be early ocular hypotony that recovers after a few hours. The eye is protected by a Cartella shield and the patient admitted for bed rest, to prevent an expulsive haemorrhage. Prophylactic

systemic antibiotics are prescribed and tetanus toxoid is given if required. A CT scan is performed to rule out intraorbital, intracranial or intraocular FBs or penetration. An ultrasound B scan is used to localize a posterior rupture/exit site. Management is by urgent primary surgical repair.

Globe rupture is a full-thickness wound of the eyewall from a blunt injury. The eye is filled with incompressible liquid and the impact causes sufficient pressure to rupture the eye at its weakest point, by an inside-out mechanism. Rupture is commonly at the impact site, or an area of corneal or scleral weakness such as an old cataract wound.

Globe rupture must be excluded by ultrasound scan in all cases of hyphaema or post-traumatic media opacity that prevents indirect ophthalmoscopy of the fundus. The eye should not be manipulated in case of extrusion of its contents and a protective Cartella eye shield is fitted. Surgical exploration and primary repair is performed if globe rupture is suspected.

Intraocular foreign bodies (IOFBs) cause 14–17% of all ocular war injuries [43,44]. They are a very important subset of eye injuries as they have a modifiable outcome using modern diagnostic, therapeutic and surgical techniques. They must be excluded in all cases of ocular blast injury. The final resting place of and damage caused by an IOFB depends on the size, the shape and the momentum of the object at the time of impact, and the site of ocular penetration [45]. In modern warfare a large proportion of IOFBs treated are from grit and stones thrown up by explosions, they are frequently multiple. Appropriate eye protection would significantly reduce the incidence of IOFB injuries [46].

Systemic and topical antibiotic therapy should be started to prevent endophthalmitis. Topical corticosteroids are used to minimize inflammation and a tetanus booster may be appropriate. Surgical removal of a posterior segment IOFB is by pars plana vitrectomy and is as an emergency if the risk of endophthalmitis is high. In warfare-related IOFB injuries approximately half will achieve a visual acuity of 6/12 or better. Poor visual outcomes and post-operative complications were related to extensive intraocular injury [47].

(e) *Retrobulbar haemorrhage*

Retrobulbar haemorrhage is an ocular emergency where bleeding into the orbital space can result in compression of the optic nerve, leading to ischaemia and blindness. Lateral canthotomy and inferior cantholysis is performed immediately to decompress the orbit and intravenous acetazolamide and intravenous mannitol is given to lower the intraocular pressure.

Figure 7. US infantryman with 'Sawfly' combat eye protection with an embedded piece of shrapnel.

(f) Tertiary blast injury

Tertiary blast injuries are from the effects of being thrown into fixed objects or structural collapse. An explosion can cause indirect ocular effects such as Purtscher's retinopathy, a sudden onset multifocal, vaso-occlusive event associated with head and chest trauma. It causes a sudden loss of vision that slowly recovers over weeks and months; its appearance is of multiple patches of superficial retinal whitening with retinal haemorrhages surrounding a hyperaemic optic nerve head.

(g) Quarternary blast injury

Quaternary blast injuries are those not caused by primary, secondary or tertiary blast. They commonly include thermal and chemical burns around the eye and adnexae.

Thermal burns are usually from contact with hot liquids, gases or molten metals. Tissue damage is typically limited to the superficial epithelium, often with damage to the corneal limbal stem cells that retards re-epithelialization. Thermal necrosis and ocular penetration can occur, especially in conjunction with IOFBs. Topical antibiotics are prescribed for epithelial defects and conjunctivitis. Initially the lid swelling may protect the corneal surface, but sloughing and contracture can lead to corneal exposure, requiring topical ocular lubricants.

Chemical burns are blinding emergencies. Alkaline agents such as lye or cement penetrate cell membranes and cause more damage than acidic agents. Acid burns are usually from battery acid explosions which precipitate on reaction with ocular proteins. Corneal epithelial defects range from superficial epitheliopathy to total epithelial loss. Limbal ischaemia is a whitened area without blood flow around the eye. The whiter the eye is, the worse the burn. Management of chemical burns is by immediate copious irrigation with Ringer's lactated solution for 30 min or an amphoteric solution such as Diphoterine (normal saline or even tap water are crude alternatives). The pH should be measured after 5 min on ceasing irrigation and irrigation continued until neutral (pH 7.0). For all severe burns, the conjunctival fornices are swept with a glass rod to

Figure 8. Plain skull X-ray demonstrating orbital foreign body (rubber bullet).

Figure 9. Computerized tomography scan of eye demonstrating IOFB in blast injury patient.

remove retained debris and break conjunctival adhesions. Topical antibiotic ointment and cycloplegic drops are prescribed along with topical and oral ascorbic acid (vitamin C) to promote fibroblast activity. Oral analgesia is used if required. Topical steroids are used cautiously as they may cause corneal melting. Amniotic membrane grafts promote corneal surface healing and are especially useful in the management of severe burns.

Sympathetic ophthalmia is a bilateral panuveitis that causes a painful red eye with visual loss in a patient with a history of uveal tissue prolapse from an eye injury. It occurs in approximately 1:500 cases of open globe injury. The injured eye is termed the exciting eye and the fellow eye is the sympathizing eye. Exposure of retinal specific proteins to the immune system during the injury of one eye causes an

Figure 10. An ultrasound biomicroscopic image showing metallic foreign body (arrow) and its shadowing effect (asterisk) in the ciliary body [54].

autoimmune reaction to both eyes at a later date. It can occur as early as 5 days and as late as 60 years after the injury, though 90 per cent of cases occur within the first year. The typical appearance is of granulomatous panuveitis with mutton-fat keratoprecipitates, usually with multiple yellow Dalen–Fuchs subretinal nodules apparent on fundoscopy.

It is commonly thought that prevention of sympathetic ophthalmia is by enucleation of blind traumatized eyes within 7 to 14 days of the injury. There is little immunological evidence to support this management or time limit. Evisceration of the globe contents rather than enucleation of a totally disrupted eye will usually give a better cosmetic result and there is no evidence to suggest that it increases the risk of endophthalmitis [48]. If it occurs, topical and systemic steroids are the mainstay of treatment.

Endophthalmitis after ocular trauma is a devastating complication of penetrating ocular trauma, particularly IOFBs. The ocular tissues are rapidly destroyed by direct invasion by the organism, its toxins or from the inflammatory response. Delay in globe repair increases the risk of endophthalmitis. It is rare after battlefield eye injuries, probably as a result of early treatment with broad-spectrum systemic antibiotics. A diagnostic tap is performed and systemic and intra-ocular antibiotics are administered. The prognosis is generally poor but can be extremely variable because of the variety of organisms involved. The visual acuity at the time of the diagnosis and the causative agent are most predictive of outcome.

Recovery from ocular blast injuries can be divided into three stages. The first stage of active general treatment and healing lasts for three weeks. Poor vision at this time does not preclude a satisfactory outcome. The second stage is from 4 to 12 weeks where individual clinical patterns requiring specific treatments are recognized. An accurate prediction of the final acuity can be made at this time; it is largely dependent on the state of the macula and if there is other chorioretinal damage. The third, delayed, stage allows very few late changes in visual acuity, though improvement is usual.

Figure 11. Blinded war veteran demonstrates the BrainPort Vision Device.

3. EYE PROTECTION

Eye protection reduces the number of eye injuries in warfare, particularly secondary blast injuries (figure 7). Cotter and La Piana analysed the data from the Vietnam War and, using a theoretical model, proposed that had the standard current US Army 2 mm thick defence goggle been worn, 52 per cent of eye injuries would have been prevented [49]. Applying this figure to the Vietnam War overall, 5000 eye injuries from US and allied forces could have been prevented by the use of eye protection [50].

Eye protection is provided to US and UK service personnel. Its uptake is variable, as combatants will often complain that eye protection degrades their vision due to misting, a poor field of view and easy scratching. Enforced use of eye protection in US military convoys in Iraq in 2004 reduced the incidence of eye injuries to 0.5 per cent from a conflict wide incidence of 6 per cent, back to the levels experienced in the Crimean War [51] (table 1).

4. MANAGEMENT OF THE INJURED EYE

Ocular trauma patients are often particularly stressed and should be made as comfortable and relaxed as possible. It is important to assess if two eyes are present and if they are grossly intact; this can be difficult and examination under anaesthetic may be required.

Meticulous note-keeping is essential as eye injuries will often require preparation of legal and insurance reports or police statements. An accurate list of all injuries to the eye, its adnexae and other injuries to the body should be made. Eye protection or eyewear used at the time of injury should be noted.

Associated cranial trauma should be considered especially if there are associated facial injuries or penetrating trauma. Subconjunctival haemorrhages with no posterior border and severe bilateral ecchymosis ‘panda eyes’ are signs of possible base of skull fractures. Facial structures are assessed for cheek swelling, flattening or asymmetry from a malar fracture. Proptosis, a bulging eye, suggests an orbital haematoma. The orbital rim is palpated for steps indicating a fracture, or crepitus indicating surgical emphysema from a fracture in an air sinus. Infraorbital

hypoesthesia indicates infraorbital nerve involvement in an orbital floor fracture.

The vision should be assessed using a Snellen chart, or estimated using typed script. If no letters can be read, the ability to count fingers, to see hand movements and to perceive light are recorded in that order as CF, HM, PL and NPL (nil perception of light), respectively. Extraocular muscle assessment is then performed in all nine positions of gaze while following a pen torch or target. Pupil examination is performed; shape, symmetry, the red reflex and pupil reactions to light are recorded.

The eyelids are examined for lacerations and everted to detect FBs. If globe rupture is suspected the lid must not be everted in case it puts pressure on the globe and causes extrusion of ocular contents. Lid wound assessment includes an estimation of the depth to assess thickness, tissue loss and lacrimal canaliculal involvement.

Globe examination is initially performed using a pen torch to examine the anterior segment, pupil reactions, lids and adnexae. An estimation of the anterior chamber depth can be made by holding the torch side on to the limbus. Pupil dilation (mydriasis) is useful for posterior segment examination, but must only be performed after the pupil reactions have been assessed, an anterior penetrating injury or globe rupture has been excluded and the anterior chamber is judged not to be shallow.

5. SPECIAL INVESTIGATIONS

A plain skull X-ray is performed to exclude cranial and facial fractures and will visualize radio-opaque FBs (figure 8). CT scans are the test of choice for orbital and IOFB localization (figure 9) [52]. A CT scan will often diagnose other unsuspected cranial and facial injuries.

Ultrasound is useful for IOFB localization and can be used even in the presence of a ruptured globe (figure 10). Magnetic resonance imaging (MRI) is contraindicated for metallic IOFB. The magnetic flux may cause them to move within the eye and cause further intraocular damage.

6. FUTURE RESEARCH

The amniotic membrane (AM), or amnion, is the innermost layer of the placenta and consists of a thick basement membrane and an avascular stromal matrix. It promotes corneal epithelialization and has anti-inflammatory activity. Preserved AM is frequently used to treat persistent corneal epithelial defects and opacities from ocular burns after explosive injuries [53]. The Academic Department of Military Surgery and Trauma (ADMST), in collaboration with the University of Nottingham, is assessing the effects of different AM preparations on epithelial cell growth using an *in vitro* model of corneal tissue. We are attempting to augment the effects of dry-preserved AM for use as a dressing after ocular burns in the deployed environment.

ADMST is creating a rat model of ocular blast injury to measure the forces transmitted to the eye

and optic nerve following blast injury. This will define the *in vivo* markers that correlate with the extent of ocular injury after trauma and the pathological processes that occur in commotio retinae and traumatic optic neuropathy. The model can then be used to test neuroprotective interventions such as immune system activation to neutralize damaging compounds and secrete neural growth factors. PN-277 and copolymer-1 (Cop-1) are both peptides which act as weak antigens thereby exciting an immune response, and have shown a retinal neuroprotective effect [54]. A dosage and regimen of vaccination will be developed for both these compounds from an animal model to develop neuroprotective vaccinations for retinal injuries in military personnel.

For soldiers who have been blinded in warfare, ADMST is helping to develop the BrainPort Vision Device (Wicab, Inc. Madison, WI), a sensory substitution device which uses the tongue as a portal for creating a sense of vision. The device uses the picture from a spectacle mounted camera to be represented on an electrotactile array that is presented to the tongue using the intact sense of touch to give a representation of the outside world to the user. The tongue is sensitive, mobile and wet, making it ideal to receive and maintain electrical contacts. Shape and pattern perception with electrical stimulation of the tongue appears to be as good as or better than with finger-tip electrotactile stimulation, and the tongue requires only about 3 per cent (5–15 V) of the voltage, and much less current (0.4–2.0 mA), than a finger-tip [55].

The BrainPort Vision Device consists of two components, a camera, a tongue array consisting of 400 gold-plated electrodes and a Controller containing a microcontroller, drive electronics, signal processors, battery, timer and user controls that connects to the Intraoral Device with a 30 × 1.2 cm ribbon cable. The subject learns to adjust the unit and understand the sensory input to the tongue in a controlled fashion (figure 11). Proper use of the device requires orientation sessions during which the patient is first taught how to use the location and pattern of the stimulus on the tongue interface to detect and identify objects. The next step is to use the device in a real-world setting to find doors, people and objects in rooms. Progress is measured using a standardized obstacle course test.

REFERENCES

- 1 Belkin, M. 1983 Ocular war injuries in the Yom Kippur war. *J. Ocul. Ther. Surg.* **2**, 40–49.
- 2 Gombos, G. M. 1969 Ocular war injuries in Jerusalem. *Am. J. Ophthalmol.* **68**, 474–478.
- 3 Negrel, A. D. & Thylefors, B. 1998 The global impact of eye injuries. *Ophthalm. Epidemiol.* **5**, 143–169. (doi:10.1076/opep.5.3.143.8364)
- 4 Carley, S. D. & Mackway-Jones, K. 1997 The casualty profile from the Manchester bombing 1996: a proposal for the construction and dissemination of casualty profiles from major incidents. *J. Accid. Emerg. Med.* **14**, 76–80. (doi:10.1136/emj.14.2.76)

- 5 Mallonee, S., Shariat, S., Stennies, G., Waxweiler, R., Hogan, D. & Jordan, F. 1996 Physical injuries and fatalities resulting from the Oklahoma City bombing. *J. Am. Med. Assoc.* **276**, 382–387. (doi:10.1001/jama.276.5.382)
- 6 Thach, A. B., Ward, T. P., Hollifield, R. D., Cockerham, K., Birdsong, R. & Kramer, K. K. 2000 Eye injuries in a terrorist bombing: Dhahran, Saudi Arabia, June 25 1996. *Ophthalmology* **107**, 844–847. (doi:10.1016/S0161-6420(00)00029-4)
- 7 Belkin, M., Treister, G. & Dotan, S. 1984 Eye injuries and ocular protection in the Lebanon War, 1982. *Israeli J. Med. Sci.* **20**, 333–338.
- 8 Steindorf, K. 1914 Die Kriegschirurgie des schorgans. *Berlin Klin. Wöschensh.* **51**, 1787–1789.
- 9 Mader, T. H., Aragonese, J. V., Chandler, A. C., Hazelhurst, J. A., Heier, J., Kingham, J. D. & Stein, E. 1993 Ocular and ocular adnexal injuries treated by United States military ophthalmologists during Operations Desert Shield and Desert Storm. *Ophthalmology* **100**, 1462–1467.
- 10 Owens, B. D., Kragh Jr, J. F., Wenke, J. C., Macaitis, J., Wade, C. E. & Holcomb, J. B. 2006 Combat wounds in operation Iraqi Freedom and Operation Enduring Freedom. *J. Trauma* **64**, 295–299. (doi:10.1097/TA.0b013e318163b875)
- 11 Benzinger, T. 1945 The physiosogical effects of blast in air and water. In *German aviation medicine in World War II*, vol. ii, 1225–1259. Washington, DC: US Government Printing Office.
- 12 Rossle, R. 1945 Pathology of blast effects. In *German aviation medicine in World War II*, vol. ii, 1259–1268. Washington, DC: US Government Printing Office.
- 13 Schardin, H. 1945 The physical principles of the effects of detonation. In *German aviation medicine in World War II*, vol. ii, 1207–1224. Washington, DC: US Government Printing Office
- 14 Hamit, H. F. 1973 Primary blast injuries. *Ind. Med.* **3**, 142.
- 15 Wharton-Young, M. 1945 Mechanics of blast injuries. *War Med.* **8**, 2.
- 16 Bellows, J. G. 1947 Ocular war injuries. *Am. J. Ophthalmol.* **30**, 309–323.
- 17 Zerihun, N. 1993 Blast injuries of the eye. *Trop. Doc.* **23**, 76–78.
- 18 Dalinchuk, M. N. & Lalzoi, M. N. 1989 Eye damage accompanying explosive mine injuries. *Voen Med. Zh.* **8**, 28–30.
- 19 Scott, G. I. & Michaelson, L. E. 1946 An analysis and follow-up of 301 cases of battle casualty injuries to eyes. *Br. J. Ophthalmol.* **30**, 42–55. (doi:10.1136/bjo.30.1.42)
- 20 Duke-Elder, S. 1954 Concussion injuries. In *Text-book of Ophthalmology*, vol VI: *Injuries*, pp. 751–961. London: Henry Kimpton.
- 21 Quere, M. A., Bouchat, J. & Cornand, G. 1969 Ocular blast injuries. *Am. J. Ophthalmol.* **67**, 64–69.
- 22 Margo, C. E. 1994 Vitreous haemorrhage. *Diagnostic problems in clinical ophthalmology*, pp. 404–415. Philadelphia, PA: Saunders.
- 23 Elmassri, A. 1970 Injuries to the posterior segment of the globe. *Bull. Ophthal. Soc. Egypt* **63**, 25–33.
- 24 Kaminski, D. E. 1943 Hole in the macula as a result of ocular contusion by firearms. *Viestnik* **22**, 25–31.
- 25 Lister, W. T. 1918 War injuries of the eye. *Lancet* **20**, 67–71.
- 26 Campbell, D. R. 1941 Ophthalmic casualties resulting from air raids. *Br. Med. J.* **28**, 966.
- 27 Cooper, G. J. Biological effects of blast. Institute of Explosives Engineers/PSBD Home Office, BCC 1993/MOD.
- 28 Beiran, I. & Miller, B. 1992 Pure ocular blast injury. *Am. J. Ophthalmol.* **114**, 504–505.
- 29 Kuhn, F., Morris, R., Witherspoon, C. D., Heimann, K., Jeffers, J. B. & Treister, G. 1996 A standardized classification of ocular trauma. *Ophthalmology* **103**, 240–243. (doi:10.1007/BF00190717)
- 30 Wilkins, R. B. & Havins, W. E. 1982 Current treatment of blow-out fractures. *Ophthalmology* **89**, 464–466.
- 31 Wilson, S. A. & Last, A. 2004 Management of corneal abrasions. *Am. Fam. Physician* **70**, 123–128.
- 32 Kaiser, P. K. & Pineda II, R. 1997 A study of topical non-steroidal anti-inflammatory drops and no pressure patching in the treatment of corneal abrasions. Corneal Abrasion Patching Study Group. *Ophthalmology* **104**, 1353–1359.
- 33 Caspar, L. 1916 Subepithelial trubungsfiguren der hornhaut, nach verletzungen. *Klin. Monatsbl. Augenkeild.* **57**, 385–390.
- 34 Monney, D. 1973 Angle recession and secondary glaucoma. *Br. J. Ophthalmol.* **57**, 608–612.
- 35 Blanton, F. M. 1968 Anterior chamber angle recession and secondary glaucoma. *Acta Ophthalmol.* **46**, 886–908.
- 36 Bleiman, B. S. & Schwartz, A. L. 1979 Paradoxical intraocular pressure response to pilocarpine. A proposed mechanism and treatment. *Arch. Ophthalmol.* **97**, 1305–1306.
- 37 McCannel, M. A. 1976 A retrievable suture idea for anterior uveal problems. *Ophthal. Surg.* **7**, 98–103.
- 38 Demeler, U. 1988 Surgical management of ocular hypotony. *Eye* **2**, 77.
- 39 Kearns, P. 1991 Traumatic hyphaema: a retrospective study of 314 cases. *Br. J. Ophthalmol.* **75**, 137–141. (doi:10.1136/bjo.75.3.137)
- 40 Lister, W. T. 1918 Hunarian lecture: pathological aspects of certain war injuries of the eye. *Lancet* **20**, 67–72.
- 41 Lewis, H., Aaberg, T. M. & Abrams, G. W. 1991 Causes of failure after initial vitreoretinal surgery for severe proliferative vitreoretinopathy. *Am. J. Ophthalmol.* **111**, 8–14.
- 42 Lewis, H. & Aaberg, T. M. 1988 Anterior proliferative vitreoretinopathy. *Am. J. Ophthalmol.* **105**, 277–284.
- 43 Mader, T. H., Carroll, R. D., Slade, C. S., George, R. K., Ritchey, J. P. & Neville, S. P. 2006 Ocular war injuries of the Iraqi insurgency January–September 2004. *Ophthalmology* **113**, 97–104. (doi:10.1016/j.ophtha.2005.07.018)
- 44 Thach, A. B. *et al.* 2008 Severe eye injuries in the War in Iraq, 2003–2005. *Ophthalmology* **115**, 377–382. (doi:10.1016/j.ophtha.2007.04.032)
- 45 Woodcock, M. G., Scott, R. A., Huntbach, J. & Kirkby, G. R. 2006 Mass and shape as factors in intraocular foreign body injuries. *Ophthalmology* **113**, 2262–2269. (doi:10.1016/j.ophtha.2006.06.002)
- 46 Thach, A. B., Ward, T. P., Dick, I. I. S. B., Bauman, W. C., Madigan, W. P., Goff, M. J. & Thorsden, J. E. 2005 Intraocular foreign body injuries during Operation Iraqi Freedom. *Ophthalmology* **112**, 1829–1833. (doi:10.1016/j.ophtha.2005.04.024)
- 47 Colyer, H. H., Weber, E. D., Weichel, E. D., Dick, J. S. B., Bower, K. S., Ward, T. P. & Haller, J. A. 2007 Delayed intraocular foreign body removal without endophthalmitis during Operations Iraqi Freedom and Enduring Freedom. *Ophthalmology* **114**, 1439–1447. (doi:10.1016/j.ophtha.2006.10.052)
- 48 du Toit, N., Motala, M. I., Richards, J., Murray, A. D. N. & Maitra, S. 2008 The risk of sympathetic ophthalmia following evisceration for penetrating eye injuries at Groote Schuur Hospital. *Br. J. Ophthalmol.* **92**, 61–63. (doi:10.1136/bjo.2007.120600)
- 49 Cotter, F. & La Piana, F. G. 1991 Eye casualty reduction for eye armour. *Mil. Med.* **156**, 126–128.

- 50 Tredici, T. J. 1968 Management of eye casualties in South East Asia. *Mil. Med.* **133**, 355–362.
- 51 Gondusky, J. S. & Reiter, M. C. 2005 Protecting military convoys in Iraq: an examination of battle injuries sustained by a mechanised battalion during operation Iraqi Freedom II. *Mil. Med.* **170**, 546–549.
- 52 Lakits, A., Prokesh, R., Scholda, C. & Bankier, A. 1999 Orbital helical computed topography in the diagnosis and management of eye trauma. *Ophthalmology* **106**, 2330–2335. (doi:10.1016/S0161-6420(99)90536-5)
- 53 Meller, D. *et al.* 2000 Amniotic membrane transplantation for acute chemical or thermal burns. *Ophthalmology* **107**, 980–989. (doi:10.1016/S0161-6420(00)00024-5)
- 54 Yang, B., Shan, L., Song, W., Shi, L. & Yuan, H. 2010 Copolymer-1 immunization reduces damage in retinal ganglion cells under high intraocular pressure through altering the expression of retinal neurotrophins. *J. Ocular Pharmacol. Therap.* **26**, 11–19. (doi:10.1089/jop.2009.0037)
- 55 Bach-y-Rita, P., Kaczmarek, K. & Meier, K. 1998 The tongue as a man–machine interface: a wireless communication system. *Proc. 1998 Int. Symp. Information Theory and its Applications, Mexico City*, pp. 79–81.