9 Biodynamic and Emergency Management of Blast Eye and Orbital Injuries

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INTRODUCTION

Conventional or nonconventional combat is ever-present around the world. Physicians whether civilian or in uniform have unnoticed weaponry biophysics and pathophysiological effect, perhaps the same applied to the medical journals? It is traditionally ignored subject probably by believing that such knowledge has no therapeutic benefit for the casualty management, especially when the country of the editor of medical journal is not at war. The new millennium technological advances, contrary to expectations, are not used for peaceful living of mankind. Presently, none of us is safe, and possibly will be for years to come. The use of large amount of conventional munitions and new high explosive material during the first decade of the first century of the new millennium is an unhappy message. This is the beginning to diagnose and manage a newly recognized type of injury which is inflicted by the blast wave alone. Comparing the advances of wars weaponries that took place in the last century, the medical profession worldwide should be prepared to face the consequences of the advances in improvised explosive devices (IEDs) expected and the widespread devastation that these and coming explosive weapons can cause.

In recent years there has been an unprecedented increase in suicides, homicides and insurgents using IEDs such as, cars, walking bombs, land mines, rocket-propelled grenades (RPG-7-29), thermobaric 'enhanced-blast explosives', explosive-formed projectiles and possibly suitcase of nuclear devices. All of these devices have been shown to dramatically increase eye and orbital injuries. Physician's knowledge of pathophysiology, biophysics and mechanism of primary and secondary blast-wave front effects as primary causative factors of war-induced eye injuries is useful for management. This review aims to reappraise eye and orbit blast injuries and their prevention and management.

Between the years 1947 and 2000, there were 280 local wars with 25 million deaths and 100 million casualties (Canadian Red Cross). 15 million of them expected to have some sort of eye or orbital injuries. Currently there are approximately 40 violent conflicts are actively going on. Blast ocular trauma causes hyphema, corneoscleral lacerations, penetrating injuries of eyeball, traumatic cataracts, retinal detachment and optic nerve injury. The ocular trauma may be associated with fracture of the bones of nose and orbit, brain damage, maxillofacial crushed fractures and multisystem injuries.

Physics of Blast Biological Effects

The molecules of gas in the atmosphere around us are in constant thermal motion. On average at sea level, there are 30 million billion molecules in every cubic millimeter of air, moving at the speed of 300 m/s and bumping into one another 100 million times each second, after traveling only 0.001 mm. This continual bombardment of gas molecules against any solid surface exerts a force on every part of that surface and is most appropriately expressed as a force per unit area, or a pressure.[1]

An explosion of solid or liquid chemical materials rapidly releases energy and produces a large volume of gaseous product. Weapons depend on high- and low-explosive potentials, thermobaric 'enhanced-blast explosives' that can produce large overpressures with long positive durations (that is, the time over which the pressure is greater than the ambient undisturbed pressure) and nuclear detonations. All provide this change in potential energy to kinetic injury in a very short period of time. The extreme compression of molecules by this change in energy creates wave front of locally high pressure, the blast wave which moves spherically outwards from the center of the blast.

Clinical findings of a victim supply us information on how the leading edge of the blast wave exercises a severe shattering and shearing from an increase in ambient pressure. The air molecules compressed to such a density that the pressure wave itself acts more like a solid object striking a tissue surface.[2]

The physical processes involved in the body's response to blast actually comprise three steps: (a) the body's external surface moves rapidly with the sudden increase in environmental pressure, (b) the air containing organs become distorted, creating stress within the organ tissues, and (c) when the stress exceeds the strength of the tissue, damage of the organ occurs.[3]

The spherical front of this blast wave exhibits a discontinuous increase in pressure, density and temperature, known as a shock front. A blast wave can inflict several types of bodily injuries through one or more of the following distinct mechanisms. These mechanisms fall into five categories, which are as follows.

(1) Primary Blast Effect on Injured Tissue

The leading edge of a blast wave, which consists of few millimeters of over pressurized air, is called the blast front and it moves rapidly in all directions from the epicenter of the explosion. The surfaces of the body that are oriented towards the blast front receive the greatest load. The geometry of surrounding structures may deflect the blast wave, or it may focus the wave, particularly inside partially open enclosures where the blast loading can be significantly higher than it would have been in a free field. In 2003, Thach reported that small changes in atmospheric pressure can lead to high-velocity winds.[4] For instance, a peak pressure of as little as 0.25 psi can generate wind speeds of up to 125 mph. Comparing these winds with the over pressurized blast wave that moves rapidly away from the center of detonation and which can be as high as 3.7–18.6 miles/s (6000– 30,000 m/s; or 13,421–67,108 mph), for high explosives and 900 ft/s (270 m/s) for low explosives, it is a blast wind (not blast wave). Stewart reported that when a high explosive detonates, it is converted almost instantaneously into a gas at a very high pressure and temperature.[5] For example, major ingredient in composition C-4 or cyclotrimethylene trinitramine (RDX) can generate an initial pressure of over 4×10^6 psi.[6] These high-pressure gases rapidly expand from the original volume and generate a marked pressure wave – the blast wave that moves outward in all directions as a thin layer of compressed air. The displaced air then compresses and forms a vacuum returning to the point of detonation (negative wave).

Temperatures from the explosive gases can reach 3000°C or more in the center zone, especially in thermobaric explosion. Victims close to the detonation can sustain third-degree burns that can be fatal, and the temperature produced by thermobaric is greater than in high explosives. For a single, sharp-rising blast wave caused by detonation of a high explosive, the damage to human structures is a function of the peak pressure and the duration of the initial positive phase. The greatest energy transfer occurs at points where tissue density changes.[7]

When a blast front reaches a victim in a spherical shape {as in improvised explosive devices (IEDs)} or in cone shape (as in some mine explosions), it causes an enormous and almost instantaneous rise in ambient pressure, filling the space with high-pressure gases in 0.001s}. Because explosive gases continue to expand from their point of origin, a longer negative under pressure (relative vacuum) follows the peak positive over pressure. Both the positive over pressure and the negative under pressure are capable of causing significant primary blast injury (Fig. 9.1).[5]

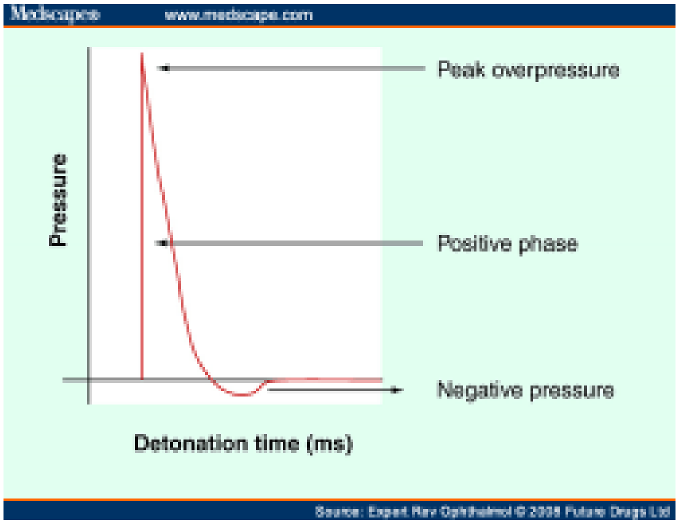

Fig. 9.1: Air blast shock wave pressure/time

The sudden pressure change caused by the blast wave can damage living tissue through four mechanisms: (1) spalling, (2) implosion, (3) acceleration–deceleration, and (4) pressure differentials. Kluger stated that an overpressure of 1.8 psi generates glass shards capable of penetrating the abdominal wall and 3 psi overpressure can throw the human body, causing fatality 1% of the time.[8] Lung injury with 1% mortality is observed at 35 psi overpressure, but results in 99% fatality at 65 psi. A charge of 25 kg TNT will induce a 150 psi peak over- pressure for 2 ms that travels at 3000–8000 m/s and more explosive will prolong the duration of the blast front, adding to the wounding potential. The blast wind movement induced by the explosion depends on the air density and the blast wind velocity; the higher the velocity the greater the generation of casualties. By single or multiple biophysics, effects of blast wave impact causes variable wound patterns and this demonstrates how a complex consequence of blast effect acts. A great deal remains to be discovered concerning how biological structures primarily respond to blasts, both in terms of responses of individual tissue and tissue microbiology to vibratory energy and why some anatomical tissues are more susceptible to primary blast than others.

The air-filled organs and air-fluid interfaces are the organs damaged by dynamic pressure changes at tissue density (i.e. air–fluid) borders due to the interaction of a high-frequency stress wave and a lower frequency shear wave. One or the other of these waves predominates, depending on the characteristics and location of the blast. Rupture of the tympanic membranes, pulmonary damage and air immobilization and rupture of hollow viscera are the most important primary forms of blast injury.[8-13] Ocular and orbital anatomical region containing liquid and other tissue media bounded by thin bone plate walls of air-containing sinuses are also vulnerable.

During a blast in the open, scaling is affected by factors such as topography and conditions of burst and energy transfer. These augment or attenuate the blast wave in an unpredictable manner. Scaling of blast intensity indoors, inside buildings or bus shelters are more lethal and have tissue destructive effects attributable to the uncertain, almost infinite possibilities that are inherent in reflection from walls of varying geometry and structure. The same effects apply to the body tissue structure and its architecture.[7,14–18] Blast injury has an overall lethality of approximately 7.8% in open air. This jumps to 49% when the blast occurs in confined spaces.[19]

2. Secondary Blast Effects

Secondary blast injury is much more common than primary blast injury. Indeed, secondary blast injury is the most common cause of death in blast victims. Up to 10% of blast survivors will have significant eye injuries.[20] Penetrating fragments made of different kinds and shapes of objects ranging from conventional shell fragments to car fragments, ground particles, sand and pebbles or other components may cause devastating damage to the eye and body.

In conventional war, the most common weapons responsible for ocular injuries are shell fragments from heavy artillery, rockets, grenades, mines and other nonmagnetic particles. Wong *et al.* reported that combat eye-penetrating foreign bodies are approximately 55% nonmagnetic, reflecting the nonferrous composition of mines and secondary missiles.[21]

In urban explosions, secondary blast causative factors are different inside populated cities. Glass fragments from windows are notorious for causing ocular injuries. They often do not kill, but can cause blindness and ruptured globes. At the speed that explosively propelled fragments of glass travel, there is no time for the blink reflex to operate.

3. Tertiary Blast Effect

Propelling of the body against walls or objects, and blunt trauma from building collapse resulting in crush injures to any part of the body including the eyes, and orbital and facial bone constitutes the tertiary blast effect.

4. Quaternary Blast Effect

Asphyxia through inhalation of fumes from toxic, burnt materials and burns by high thermal explosive on the cornea are the key factors in the production of the quaternary blast effect.

5. Quinary Blast Effect

The quinary blast effect includes the toxic substances that are absorbed through wounds or by inhalation.[9,21]

Relevant Orbital and Ocular Anatomy

The orbit encloses vital anatomical organs such as eyeball, muscles, fat, vessels, and nerves in a thin boney cavity. An air-fluid-soft tissue medium forms a unique interface for a shock wave to spread through to the surrounding medium related to the eye globe and orbital tissues. The unique eye position due to the architecture of the middle part of the facial skeleton extends laterally and thus exposes the surface of the eye and orbit to the blast injury. Multiple biophysics effects of blast waves demonstrate how blast affects individual tissues as a result of their vibratory energy and why some anatomical tissues are more susceptible to primary blast. The interaction of a high frequency stress wave and a lower frequency shear wave determines the degree to which organs are damaged by dynamic pressure changes at tissue-density borders.

The orbital walls consist of thin plates that separate orbital tissue content from the CSF and brain superiorly, the paranasal sinuses medially and inferiorly, and the lateral plates from the temporal region. Only the circumferential orbital rim is a thick resilient bone. The lateral wall has the lowest frequency of injury of all civilian facial fractures. The medial wall is of critical importance during blast implosion of the ethmoidal air cells in the medial wall, as it is composed of thin bones ranging from 0.2 to 0.4 mm in thickness close to the thin cribriform plate that leads to crush injury by blast effect into nasal-orbital-ethmoidal (NOE) comminuted fractures. The superior wall is moderately resistant to fracture; the orbital roof is 3 mm thick in the posterior and is thinnest just behind the superior rim in the anterior. The inferior wall is the most vulnerable to injury.

Orbital volume is approximately 35 ml, of which only 7 ml (20%) is occupied by the eyeball. The remaining 28 ml contains muscles, nerves, fat, glands and blood vessels.[22,23] The orbit can thus have room for a compressible mechanism and accommodate a relatively large foreign body without showing great disturbance of function.[24] The distance from the eyeball to the medial orbital wall (6.5 mm) is slightly larger than the distance to the lateral wall (4.5 mm).[20] Medially penetrating objects will injure the eyeball less frequently than an object entering laterally.[25]

Experimental Analysis of Dynamic Stress and Strain Properties of Eyeball Rupture

Ocular globe rupture may occur when a primary wave impacts the orbit causing anterior-posterior compression of the globe and raising intraocular pressure to a point that the sclera tears. A rupture also may occur when the integrity of the outer membranes of the eye is disrupted by a blast over pressure wave, penetrating shell fragment, sand particles or dirt that cause abrasion or deep laceration which weaken the eye wall. Propelling of the body against walls or objects or otherwise may lead to eye and orbital injuries when it is hit by protruded blunt or penetrating object as a tertiary blast effect.[26]

Biophysics explains the pathophysiology of why secondary blast explosively propelled fragments inflict eye and orbital injury as they penetrate or rupture the eyeball. According to the supercomputer simulation study by Uchio *et al.*[27] the sizes of missile above which corneal rupture occurred at velocities of 30 and 60 m/s were 1.95 and 0.82 mm, respectively. However, the missile sizes causing sclera rupture were 0.95 and 0.75 mm, at velocities of 30 and 60 m/s, respectively.

As a correlate, another pressure system study was conducted to examine the static and dynamic properties of healthy postmortem human eye rupture and pressures needed. It wound help to determine the primary blast wave front stress force that is required to inflict eye and orbital injury and conversely, the protection needed to prevent it. Voorhies in her thesis submitted to the Faculty of the Virginia Polytechnic, found that maximum rupture stress for the somewhat static tests was found to be 11.17 MPa for human tissue, whereas, stress for the dynamic tests were found to be 30.18 MPa. Maximum rupture stress results correlate well with static material properties used in the published research, such as 9.4 MPa and dynamic properties of 23 MPa.[28]

Eye ruptures in the region of the limbus are often circumferentially oriented, and ruptures at the equator are often zonally oriented. This reveals the predominant direction of collagen fibers in those two regions, which is circumferential at the limbus and meridional at the equator.[29–31]

The Blast Wave Front and Facial Morphological and Anatomical Peculiarities

Head has an external morphological contour ball-shaped, the facial segment is an irregular part of it, with anatomical soft and hard tissue variations. The lateral morphology differs from anterio-posterior or posterio-anterior and more important is the presence of air containing sinuses in the middle-third and the mandible. Spherical blast wave injuries inflicted to maxillofacial region are different from those inflicted to extremities, lungs and brain. When the middle- third of facial skeleton is compressed; the convexity of the wave creates a concave depression in the previously convex structure in the implosion phase. It is followed by miniature re-explosion of compressed air sinuses that leads to egg-shell crushed injury with soft tissue shredding. The blast wave itself may cause comminuted fractures in the sinus walls without leaving any external laceration. These are much like crystal ball crushed splinter to fragments, with exception that the bone fragments may remain attached to the periosteum and preserve most of the architecture despite shape disruption.

Effects of Face Positioning and Eye Injuries

Personnel oriented face-on to the blast wave offer little resistance to the dynamic pressure component of the wave especially in the nasal-orbital-ethmoidal (NOE) region which contains multiple air sinuses and thin bone plates. The occipital orientation gives better eye protection against the blast wind. For persons side-on to the approaching blast wave, the dynamic pressure and incident pressure are additive in the region. In the short-duration blast waves (a positive phase of 3 msec or less), there is a twofold difference in magnitude between the curves for the end-on and side-on orientations. Individuals positioned against a large reflecting surface and normal to the incident shock would have the peak reflected pressure as their effective blast dose.[32]

Blast Mechanism and Most Common Causes of Middle-third of Facial Skeleton and Ocular Injuries

Surgeons, physicians as well as ophthalmologists should have interest in the pathophysiological effects of explosive devices on the human body in general and eye and orbit in particular. Nasal-orbital-ethmoidal skeleton injuries due to primary blast wave dynamic impact with sufficient uniformed force leads to a total collapse of the tensile strength gained by architectural features and bone buttresses of the paranasal sinuses and orbital region. When the skeleton is compressed it will be fractured to small, thin bone fragments. The convexity of the wave creates a concave depression in the convex facial skeleton in the implosion phase. This is followed by an explosion phase, which brings it back to somewhat the original shape. The implosion and miniature re-explosion of compressed air sinuses leads to crush injury to this region. The primary blast effect shock wave propagates through the different medium of eye globe and

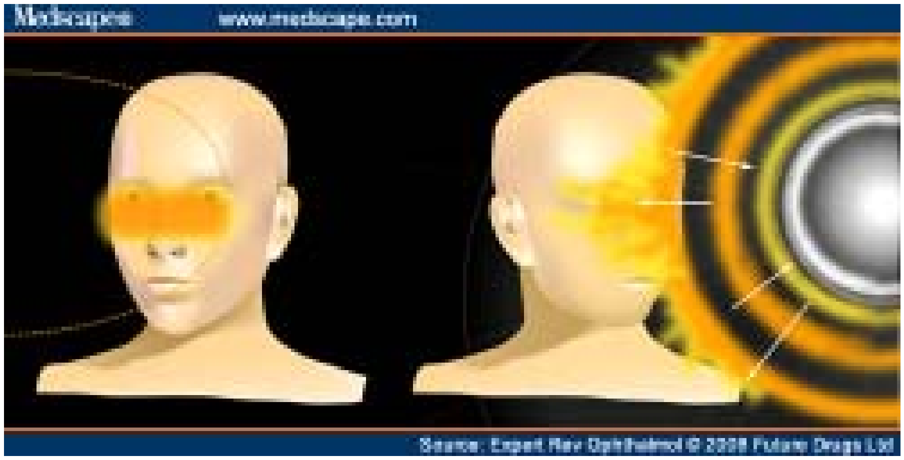

Fig. 9.2: Physical effect of the spherical blast wave front smashing the middle-third skeleton

related orbital anatomical tissues causing contusion and concussion to organs and tissue in the region. It may also affect brain tissue by transverse wave propagation through orbital roof, damaging the cribriform plate, olfactory plate, and cause frontal and ethmoidal sinuses fractures. This is similar to a crushed eggshell by blast wave impacts to middle-third facial skeleton; this could be anterior or unilateral to this region[33,34] (Fig. 9. 2).

Eye Injury

The secondary blast effect plays a major role in ocular injuries from fragments of projectiles such as shrapnel, pebbles, and fragments of glass and sand particles with environmental debris in and outside the area of detonation. Particle impact on the sclerocorneal membrane creates processes such as stress mechanical contact. The inertial and stress concentration effects in these situations often produce partial or complete corneoscleral contusion and laceration, which diminishes ocular wall resistance. The primary blast wave impacts on the weakened eye wall, facilitating a rupturing of the eyeball through the mechanism of spalling and pressure differentials of different eye and orbital medium. Sclerocorneal abrasion or partial or almost full thickness laceration that weakens the eye elastic collagen wall increases the possibility of rupture by lower primary blast wave stress impact.[35] The primary blast wave front impacts to the orbital region due to spalling and to pressure differentials and the eye globe sustained stress of high pressure force may be enough to rupture the eyeball.[36,37]

When the orbital region surrounded by air-filled sinus is exposed to the blast wave, air emboli may be seen in the retinal vessels on funduscopy. A careful neurological assessment is important, bearing in mind that the neurological state may deteriorate rapidly due to air embolism.[38,39]

Improvised Explosive Devices and Eye and Orbital Blast Injury Patterns

Improvised explosive devices as well as explosives in general have the potential to cause life-threatening multisystem injuries and mass casualty, one of these vulnerable systems to blast is the eye and orbit. The pattern of injuries occurs due to the explosive composition and type of explosive and carrier/container delivery method. Also, the distance between the victim and the blast epicenter, whether the blast occurred in a closed or open space, and any surrounding environmental barriers or hazards may be contributing factors. The major blast weaponry that leads to eye injuries includes IEDs, mines, rocket-propelled grenades (RPG-7 and more powerful RPG-29), conventional war bombs, enhanced-blast explosives as thermobaric and explosive formed projectiles (EFPs).

In its annual global survey of terrorism, the US State department said that 14,338 attacks took place in 2006, mainly in Iraq and Afghanistan. The report stated that 6600 (45%) of the attacks, took place in Iraq, killing approximately 13,000 people (65%) of the worldwide total of terrorist-related deaths in 2006.

Mader *et al.* prospectively examined severe ocular and ocular adnexal injuries that were treated at the 31st Combat Support Hospital during the portion of the Iraqi insurgency that took place from 20 January to 12 September 2004.[40]They mainly studied the incidence and characteristics of ocular and the adnexal injuries. The results showed that 207 patients suffered severe eye and adnexal injuries, including 132 open globes. Blast fragmentation from munitions caused 82% of all injuries. The most common single cause of injury was the IED, which caused 51% of all injuries. Enucleation was performed in 41 eyes, of these 24 were traumatized by IEDs.

In a study carried out on ocular injuries in survivors of IED attacks on a commuter train, Mehta *et al.* found that 16 of 28 patients (57.1%) had ocular injuries whereas 12 (42.8%) were found to be normal.[41] Injuries were seen unilaterally in 10 patients and bilaterally in 6, yielding a total of 22 injured eyes. The common injuries were periorbital hemorrhage (9 eyes; 40%); first or second-degree burns to the upper or the lower lid (7 eyes; 31.8%) and corneal injuries (8 eyes; 36.3%). Open globe injuries were seen 2 eyes of 2 patients (9%). One patient (4.5%) had a traumatic optic neuropathy.

These two studies indicate the secondary blast effect on the eye and orbit is higher in the outdoor than indoor mainly due to flying debris. In a commuter train the expected glass fragmentations would travel outside owing to the primary blast wave.

Triage of Patients with Eye Injuries

Screening of wounded or injured in the war or disaster is important mainly to determine priority need and ensure efficient use of medical and surgical manpower. On the detonation site or at the battlefield, the victim should be examined for evidence of obvious signs and symptoms of serious blast injuries, such as pulmonary, head or extremity amputation. If a hidden injury to another organ is suspected, the victim should be referred to a trauma center for initial triage, before concentrating on the eye. In severe injuries, basic and advanced life support is the primary objective until the patient is stabilized. A consultation for visual system evaluation should be prompt and must be triaged among the multiple consultations and ongoing critical care needs required for the individual patient. If neurosurgical concerns prevent pupillary dilation, a comprehensive evaluation of the retina, choroids, and optic nerve head may be compromised.

The eyes should be thoroughly checked by examining pupillary reactions, ocular movements, gross deformities, globe perforations, enophthalmos, exophthalmos and displacement of globe. After examination the injured eye should be covered immediately by a simple eye patch for protection. No pressure is to be placed on the open globe. Victims with direct eye injury, not associated with other serious head, facial or general injuries, should be immediately referred to the ophthalmology department.

Emergency Identification of Severe Eye Injuries

When time is critical due to handling mass casualties and multisystem major injuries, the primary or secondary eye and orbital injuries are unfortunately likely to remain untreated because of life-saving priorities. In the case of a continuous flow of new casualties in long-standing front-line battle or more than one explosion in one city that exhausts medical resources, eye injuries should not be missed. In a study to determine the type and frequency of ocular injuries in patients with major trauma, 16% of the cohort had ocular or orbital trauma. Of patients with injuries involving the face, 55% had ocular or orbital injuries.[42] A gross assessment of the visual acuity should be obtained with the use of light and counting fingers. 'Near charts' are also helpful for the trauma patient. Therefore, the patient should be examined and primary diagnosis determined. When the mechanism of injuries involves small fast moving objects, such as those that occur in blast or other explosive injuries, eyes should be checked in triage and emergency units by well-trained staff. However, in certain circumstances emergency room personnel are not properly trained to manage the ocular blast injuries. The eye should be routinely re-examined when the surgeon screens the blast injury patient from head to toe prior to anesthesia. The vision may be compared between the injured eye and the uninjured eye. Profound vision loss in the eye is a strong indicator of serious injury.

Nasal-Orbital-Ethmoidal Blast Injury: Management

Blast leads to a complicated compression of the middle-third facial skeleton. These cases are more difficult to treat than fractures in civilians, such as those due to direct blow or car accident injuries, which are often segmental and involve the suture-lines. Reposition by a direct or indirect approach, simple intraosseous wiring or mini plate fixation are enough for proper fixation. The crushed eggshell type of bone fragments requires restoration to the anatomic structure of the NOE to its natural and aesthetic form with preservation of function. Accurate realignment and replacement of the components should be carried out. A fundamental appreciation of the orbital anatomy can be achieved by molding, mounting and stabilization by intranasal arc scaffold techniques. However, the author's experience on blast middle facial skeleton injury found that instead of focusing on rigid fixation, the basic tenets that provide the guidelines for the reconstruction in civilian type of injuries are not always practical in blast compressed injury. Coronal or local flap exploration of the blast-crushed NOE region with impaired blood supply and fragmented bone mostly attached to its periostium is not the best surgical choice.

Repair of Blast Eye and Orbital Injuries

Blast eye/orbital injuries can be repaired to a great extent cosmetically. A 28-year-old patient sustained severe blast facial injury. Fortunately, the eyes were intact and other associated injuries were not serious. Chest radiography revealed no injury to lungs. The occipitomental view radiographs revealed crushed eggshell injuries to the NOE region, and the lateral radiograph showed destruction of tissues with no fracture lines as usually seen in civilian trauma cases. Under general anesthesia via oral intubations the wound debridement was carried out using normal saline to the pulverized bones and shredding soft tissue layer by layer, and detached devitalized tissue was removed. Small finger palpation of the deep anatomical nasal cavity through the laceration was used. When

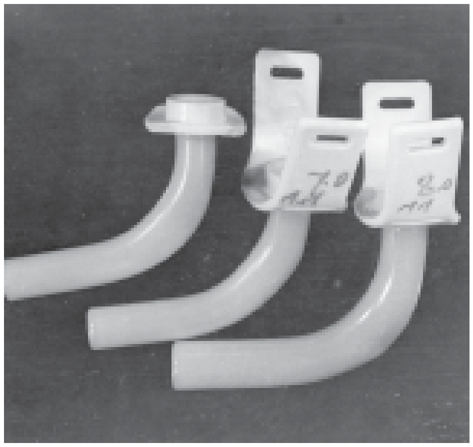

Fig. 9.3: Portex trachesotomy tubes showing convex curvature of its arch are heated in a flame, the upper curvature squeezed with straight artery forceps. Used as scaffold and physiological air path

Fig. 9.4: Number 7 and 8 tracheostomy tubes used in the treatment of NOE crushed blast injuries

it is not feasible to see the damaged nasopharynx, finger-touch feeling is used to detect lacerated surface and mucosal surface, and restore the mucosa to its proper position by the finger. Then, two Haworth periosteal elevators (Figs 9. 5 to 9.10) were inserted along the side of the finger in position to maintain mucosa in position and prevent it from collapsing until the modified portex tracheotomy tube number 8 (Fig. 9.3A) was inserted between the two elevators, then the elevators were removed. This technique was invented by Shuker in1988 for repairing the severe blast injuries of NOE region.[43] Reconstruction of hard and soft tissue can be performed over the tubes, which act as a parabolic arc scaffold. The canthal ligament is explored and is reattached to an acceptable position with the help of a 30-gauge steel wire. In case no bone segment is left attached to the ligament, then pass the silk needle through the medial canthal tendon.[44–47] Care should be taken not to pass the silk needle with wire through the portex tube as the wire will be left in position permanently while the tube will be removed.

Tubes secured on the ala of the nose, and left in position for 1 month, resulted in a cosmetically acceptable NOE region, normal phonation, physiological inspiration and expiration and no diplopia or any other complications, leading to an uneventful recovery

In the author's experience, mini-plates, which are used successfully in severe road traffic maxillofacial injuries and allow fixation of larger segments of bone fragments, were not practical in the immediate

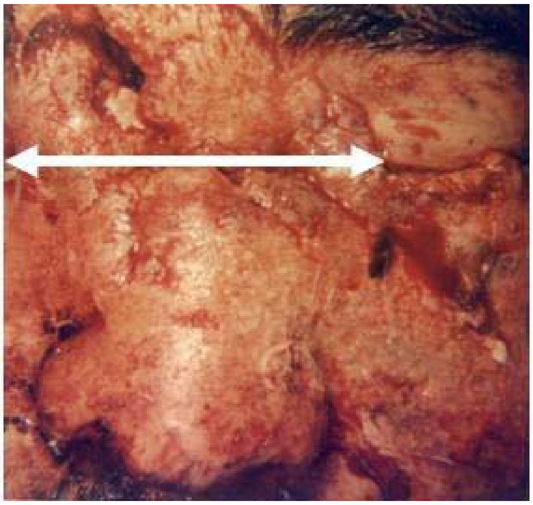

Fig. 9.5: Severe NOE blast injury more than 6 cm intercanthal space is pulverized, tethered and shredded

Fig. 9.6: Skull lateral view radiograph showing total destruction of the nasal orbital and ethmoidal region with retained multiple metallic small foreign bodies

Fig. 9.7: Portex tracheostomy tubes in position (arrow)

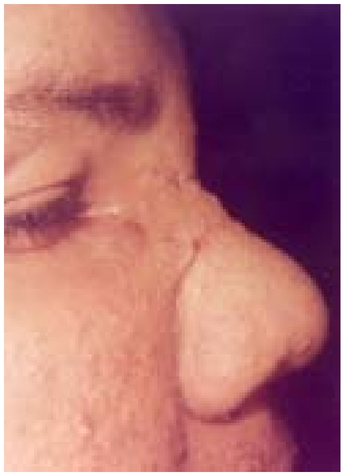

Fig. 9.8: Occipitomental skull view demonstrating the portex tube in postion

Fig. 9.9: Follow-up lateral photograph showing uneventful recovery in NOE region

Fig. 9.10: Follow-up photograph showing excellent result of the NOE region, no collapse of nasal saddle is seen

management of severe blast injuries because of small bone fragments, soft tissue mutilation, diminished blood supply and because they are time consuming at a critical stage.[48]

Severity of Ocular Injuries

The severity of blast injury of the eye may vary to a great extent from mild to very severe perforation of the globe or even avulsion of the globe. A patient of 32 years of age sustained a shell fragment injury that led to a severe rupture of the eyeball. The shell entered right medial orbital wall and exited from the left orbit, causing a ruptured eyeball and mutilation to the left medial orbital wall, as well as laceration of the eyelids. More small penetrating shell fragments peppered the face and upper arms, but no major injuries were noticed. The patient's general condition was good.

Clinical examination of the left eye showed the presence of dark uveal tissue in the socket, and a very wide intercanthal space (about 5 cm wide) due to severe destruction of the left medial orbital wall. However, no bone fragment was found in the region and there was no sign of rhinorrhea.

Under general anesthesia via oral intubation, wound debridement was carried out using normal saline. The ophthalmologist carried out enucleation of the tethered eye tissue. Reconstruction of the NOE fractures was carried out, using intra nasal stabilization portex tracheotomy tubes, as described above. The canthal ligament was explored and because fine stainless steel wires were unavailable, nylon silk was used to approximate the canthal ligament to an acceptable position. Two tracheotomy portex tubes wings were used after trimming to right shape as splint plates to secure the NOE region. The portex tubes were left in position intranasally for 1 month. The patient had an uneventful recovery (Figs 9.11 and 9.12).

The patient shown in figure 9.5 suffered blunt blast injury although there were no peppered penetrating injuries in the face and ocular region from blast particles because the patient was exposed to the indoors blast effect. This resulted in a bilateral circumorbital ecchymosis, subconjunctival hemorrhage, hyphema, vitreous hemorrhage and nasal bleeding. The management of hyphema is simple, and more often than not, the blood is reabsorbed over a period of days to weeks. However, during this time, the ophthalmologist should carefully monitor the

Fig. 9.11: Ruptured left eyeball with prolapsed uveal tissue and destroyed intercanthal space caused by shell injury

Fig. 9.12: Showing enucleated left eye and two portex tubes used for intranasal stabilization

Fig. 9.13: Bilateral massive subconjunctival hemorrhage; it was associated with vitreous hemorrhage

intraocular pressure to prevent development of secondary glaucoma (Fig. 9.13).

The presence of proptosis may indicate a retrobulbar hemorrhage. Pupillary distortion may be associated with an open globe injury. Injury to extraocular muscles, orbital fractures and orbital hemorrhage may cause motility disorders.

Avulsion of the Eye Globe

Avulsion of the eye globe is rare in the civilian injuries but occasionally it may occur in blast injury. An interesting case is being described here.

Fig. 9.14: Showing avulsed left eye with multiple facial shell fragments

A 30-year-old patient sustained severe multiple maxillofacial, orbital and neck secondary blast injuries due to an artillery bomb explosion. Shell fragment entrance from the left maxilla, transmaxillary sinus led to fractured left side infra-orbital rim and floor settled in the retrobulbar region. The left globe was avulsed, almost out of the orbital socket anterior to the eyelids, with obvious avulsion of the superior, inferior and lateral rectus muscles. The eyeball was hanging by a small attachment of medial rectus muscle. A severed optic nerve, with its free end lying within the orbit, and all the blood vessels in the region were cut off, blocked by pressure and possibly cauterized by the shell fragment thermal effect.

The eyeball was removed and the shell fragment delivered from the retrobulbar region through a direct orbital approach. The eye socket packed with vaseline gauze, and the fractured maxilla reduced to its position using direct intra-osseous wiring and upper arch bar for the upper jaw as well as exploration of the neck wound (Fig. 9.14).

The eye is subject to all the types of injuries described above, but the most common and devastating ocular injury results from the missiles created by a blast (i.e. secondary blast injury). Just as with wartime ocular injuries, those associated with terrorist blasts are most commonly due to fragments that damage the eye.[4,49–51] Although the number of ocular penetrating injuries is high due to conventional combat shell fragments, the number of penetrating eye injuries due to IEDs is far higher (and we should expect this to continue to increase) as the rate of attacks rises in high-risk populated areas.

The blast facial injury that led to a crushed eggshell NOE injury can also cause penetrating injury of the eye. The victims often present with eye pain, foreign body sensation, periorbital ecchymosis, corneal haze,

Fig. 9.15: Penetrating eye blast injury with nasal-orbital-ethmoidal region fractures

periocular contusions and decreased visual acuity. In suspected cases of open globe injury, direct manual globe pressure palpation is contraindicated. Unfortunately, ophthalmologists in front-line hospitals in most of the third-world local wars lack proper microsurgical equipments, experience in vitreoretinal surgery and expertise. Patients receive appropriate antibiotics and are evacuated to the military base hospital for management. The time taken in evacuation may jeopardize the visual recovery, if not in all, certainly in some cases. The ophthalmic specialist may be surprised as to why these sophisticated instruments are unavailable. The modern vitreoretinal techniques have improved and decreased enucleation rates for severely traumatized eyes due to blast injuries. However, many war-torn or terrorist-ridden countries would need decades to achieve the facilities available to the Western armies. Furthermore, the chances are minimal that these countries will have the budget that can afford the same standard of treatment and expertise available to the US army or NATO armies.

The NOE injury was treated by using portex tracheotomy nasal tubes and the intercanthal space approach (Fig. 9.15).

Orbital Emphysema (Tension Pneumo-orbitus)

It is common for blast injury casualties to sustain pulmonary injuries with spontaneous pneumomediastinum which presents with various manifestations. However, it is extremely rare for head and neck signs to be the sole presenting feature due to civilian chest trauma with facial subcutaneous emphysema. Air from the mediastinum can easily ascend along fascial planes and into the subcutaneous spaces of the head and neck.[52,53]

Orbital emphysema due to orbital trauma is a well-known occurrence. Visual loss due to orbital emphysema, however, is an uncommon phenomenon. An over pressurized blast wave front impact leads to implosion of the paranasal air cells; these can allow the passage of air into the orbit space and orbital soft tissues, particularly of the medial orbital wall. The orbital emphysema does not last as it deflates through the fractured orbital walls.

The compartmentalized orbital space holds the compressed air and occasionally creates a one-way valve effect that entraps this air within, as in civilian conditions, or the pressurized air dissected from oropharynx or upper part of pulmonary system. This situation can precipitate proptosis of the globe, elevation of the intraorbital and intraocular pressure, and vascular insufficiency of the optic nerve and retina. The orbit, therefore, follows pressure-volume dynamics, with a pathophysiology, in which increased tissue pressures in an enclosed space are associated with decreased blood perfusion. When the pressure within the orbital compartment, exceeds central retinal artery pressure, ischemia results from insufficient blood supply. It is more serious in retrobulbar hematoma, as the pressure effect of fluid is higher than more diffusible air pressure in tissue anatomical spaces, hematoma can cause a substantial rise in pressure if not treated and may result in blindness if not decompressed by drainage.

Irreversible optic nerve pathology may occur within 90-120 minutes of ischemia. Front-line surgeons should be familiar with retrobulbar emergent decompression by lateral canthotomy and inferior cantholysis. Medial and lateral canthal tendons limit the forward movement of the globe. Canthotomy may compensate for small increases in orbital volume by forward movement of the globe.[54-56]

Facial emphysema and orbital emphysema due to blast is mostly due to the rupture of the upper part of trachea, or marginal pulmonary alveoli. The air ascends along the mediastinum toward the subcutaneous space of the neck, causing cervico-facial subcutaneous emphysema in 70-90% of cases.[57-60]

Shuker *et al* reported pneumomediastinum and cervical emphysema subsequent to mandibular injury associated with a flare pistol shot blast injury.[61]

Suspected globe rupture is a contraindication to lateral orbital canthotomy. Signs of globe rupture include: hyphema, a peaked, teardropshaped, or otherwise irregularly-shaped pupil, exposed uveal tissue, which appears reddish-brown; and restriction of extraocular movements; a restriction that is greatest in the direction of the rupture. Subtle signs of globe rupture include subconjunctival hemorrhage, enophthalmos or a conjunctival laceration.

Orbital Emphysema Subsequent to Blast

A patient was received in the maxillofacial department with cyanosis of his lips, nails and pale cold skin and dyspnoea due to oropharyngeal emphysema. The patient's family claims that the patient was exposed to an explosion that took place in a room close to the victim's room. Owing to the patient's serious general condition and breathing difficulties, an emergency trecheostomy was decided upon and performed. Air bubbles started escaping from the surrounding subcutaneous tissue of the neck tracheostomy wound as well as from deep neck anatomical spaces in which decompression occurred. Facial emphysema was deflated subcutaneously using a large hypodermic needle, as well as eyelids emphysema deflated by a few pricks by large hypodermic needle.

The orbital emphysema decompression was attained by performing a small orbitotomy via lateral orbital rim small incision on zygomatico-frontal suture region and dissecting, carefully, the periosteum from the lateral wall to avoid traumatizing the eyeball by blunt dissection. The scissors were advanced posteromedially behind the globe, with closed tips to prevent inadvertent injury. The tips were spread gently, creating a small opening in periosteom, which was enough to allow the air bubbles out. Retrobulbar and orbital blast emphysema management is easier than hematoma and hemorrhage in the retrobulbar region. Once air under pressure is released, its effect on the blood supply will decrease. The patient had an uneventful recovery without optic nerve changes. Orbital canthotomy or inferior cantholysis could be the alternative when the pressure tension is high and expected to last longer (Figs 9.16, 9.17, 9.18 and 9.19).

Mine Eye Blast Injuries

Mines continue to injure civilians and soldiers not only during conflict but long after a ceasefire; with no differentiation between a child and a soldier.[62-65] A study of 84 patients aged 19–56 years who sustained mine blast injuries during mine clearing operations in Afghanistan from November 1992 to January 1996 was carried out by Muzaffar *et al.* in 2000.[66] It was observed that 51 out of 84 patients (60.7%) had sustained ocular trauma of a variable degree as a result of the blasts. A total of 91 eyes from 51 patients (89.2%) had been damaged. Bilateralism of damage

Fig.9.16: Severe emphysema of the anterior and posterior neck and face due to primary blast injury

Fig. 17. Severe subcutaneous and circumorbital soft tissue emphysema

Fig. 9.18

Fig. 9.19

Figs 9.18 and 9.19: showing uneventful recovery 10 days postoperatively was seen in 40 (78.4%) patients. In total, 34 eyes (37.3%) became completely blind. The prevalence of blindness caused by mine blast injuries is very high. A case of child blinded by the mine blast injury is cited below:

A 3-year-old child sustained left orbital injury due to a small plastic mine explosion (Figs 9.20, 9.21 and 9.22). There was a total loss of his left eye and orbital tissues and comminuted fractures of almost all the orbital walls, as well as severe burn to the regional tissue and severe lower extremities injuries. The general condition of the patient was stable. Under general anesthesia, orbital cavity debridement was carried out, as all the soft orbital tissue including the eyeball was lost and the bone was denuded, smoked covered by burned necrotic tissue, which was removed. Curates and periosteum elevator was used to curettage the unhealthy soft tissue. The unhealthy bone was either nibbled or decorticated using round burs deep enough until the healthy bone was reached. Fractured supraorbital rim reduction was carried out using intraosseous stainless steel wiring. No local or distant flaps were used immediately to cover the denuded bone. The orbital cavity was packed with iodoform gauze saturated with iodoform paste and was changed every 4–5 days. This was continued until secondary intention healing took place as a healthy socket especially in exposed fragmented orbital walls bone. After healthy granulation tissue filled the eye socket, local flaps from the cheek's healthy tissue infraorbital region were utilized for reconstruction successfully.

Fig. 9.20: 3 year- old-child showing left eye and orbital injury with retained FB (arrow) due to small plastic mine explosion

Fig. 9.21. Fractured superior orbital rim, loss of orbital content and burned circumorbital soft tissue

Fig. 9.22: Two months postoperatively revealed healthy healing in the left orbital region

Rocket-propelled Grenade (RPG-7 and -29) Blast Injuries

In the present era and for the coming decades, the RPG-7 – RPG-29 and its growing family have and will be the second option to IEDs for many terrorists and insurgences. It is apparent that this will be the case for years to come.

This weapon, originally developed by Bazalt, was a direct result of state research and productive enterprise in the USSR. In 1961, the Soviet army fully adopted this weapon by the end of the cold war.[67]

The RPG-7s are now so economical that they can be bought for Iesser than the price of a M.16 rifle. The weight of an RPG-7 in the firing position is 6.3 kg; it has a range of 330 m. Depending on the type of ammunition fired, a single RPG-7 round can shred a tank 3 football fields away or bring down a helicopter.[68]

Fig. 9.23: Rocket-propelled grenade-7 and -29 mechanisms showing the steps of perforating tanks armor

Shuker described the pathophysiological effect of the rocket-propelled grenade (RPG-7) and its blast maxillofacial injuries management.[34] This weapon was widely and effectively used by the Iranian troops and their militia during the Iraq–Iran War of 1980–1988. Worldwide, it is available and cheap. In the recent Iraqi and Afghanistanian conflicts, a third of the US casualties were due to RPG-7 and other versions of the RPG family. Since at least 22,834 USA troops have been wounded in action, one can easily estimate how many soldiers having lost their vision from the explosion of this weapon. This is because antitank weapons contain more explosive material and provide a greater primary blast effect. This may cause catastrophic wounds to soldiers who are hit. It causes eye injuries because of the effect of enormous secondary fragments and the primary blast wave. It can also lead to a shower spray of small, irregularly-shaped, very hot, metallic fragments at metal melting point inside and out of the compartment. This is a main causative factor of eye injury. Recently, the most advanced model of RPG-29 is used. When it strikes, the blast wave is followed by the second explosion blast peppered melt spall metal of the armor wall, which is the main cause of eye penetrating injury by small metal fragments, blast wave and thermal effects (Fig. 9.23).

Rocket-propelled grenade-7 and -29 mechanism

An interesting case of RPG-7 injury should be of some interest. A 35-year-old -soldier standing on tank turret sustained RPG-7 blast injury. On examination, there were signs of cyanosis on his lips and nails, pale cold skin, dyspnoea, pneumothorax, hemothorax and pneumomediastinum. An immediate oro-tracheal intubation and oxygen

Fig. 9.24: Rocket-propelled grenade-7 blast facial injuries, bilateral ruptured eyes, thermal

Fig. 9.25: In spite of treatment patient lost both eyes

(not under pressure) was used. He had numerous facial lacerations, burned facial skin, eyebrows and lashes in addition to an open globe injury that showed numerous orbital small metallic fragments. Chest lateral and postero-anterior radiograph revealed a collapsed left lung that was treated by chest tube.

Orbital ecchymosis and multiple penetrating facial injuries were caused by spall metal fragments. Thermal burn injury usually results from contact with hot gases leading to cell death and thermal necrosis and penetration, which can affect a large ocular surface area. Because of the very high temperature from enhanced-blast explosives more ocular injuries are expected especially when eye visor protections are not used (Figs 9.24 and 9.25).

Prophylaxis

Eyes comprise as little as 0.27% of the total body surface area and only 0.1% of the erect frontal profile. However, injuries to the eye are found in 10–13% of all combat casualties and among civilians having unexpected IED injury, which has recently increased dramatically.[40]

Cotter and La Piana studied data from the Vietnam War using theoretical analysis and found that the standard current US Army 2-mm thick defense goggle if worn would have prevented 52% of eye injuries.[69] Projecting this figure to the Vietnam War overall, 5000 eye injuries from USA and Allied forces would have been prevented.[70,71] That may also apply to the recent civilian and military eye injuries suffered from blast wave effect in undeveloped countries. Proper prophylaxis and improvements in ophthalmic care in the recent years offer hope that serious ocular injuries and their blinding sequelae will be less common in the future wars.

Wearing a helmet with a visor designed to break the stress of the wave pressure will provide protection against blast-induced injuries, especially the upper-middle-third of facial skeleton. The helmet protects the ocular–orbital region and prevents the traumatic brain injury. The use of visor can save many eyes from blast injuries. The author recommends that not only those in uniforms but civilians in unstable regions must use the visor. Better body armor and visor (eye armor) designs will help break the stress of the wave pressure and provide protection against blast-induced injuries.

Blast Eye Injuries and Recent Developments

Many thousands of people have been losing their vision due to penetrating eye injuries because modern vitreoretinal surgical techniques are not being performed when needed. Blast injury management in general and eye and orbital blast injuries specifically should need more attention and interest among ophthalmologists and other related specialties.[35]

The likelihood of a person's vision being restored has a direct relation to the immediate management of the eye injury. If the eye is significantly damaged as a result of an initial blast wave, such that it cannot be properly repaired, primary enucleation may be a reasonable option. Another instance where enucleation is indicated when the patient with a significant injury cannot come for follow-up to rule out the development of sympathetic ophthalmia.

The introduction of pars plana vitrectomy is a safe procedure for the removal of intraocular foreign bodies (IOFBs) and managing endophthalmitis. In blast wave-related penetrating injuries, foreign bodies are smaller than the conventional war missile and there appears to be a trend toward fewer ocular enucleations. Recently modern vitreoretinal techniques have been used to manage penetrating war injuries with retained IOFBs. The rate of enucleation for severely traumatized eyes with blast injuries has significantly reduced.[72-74] The percentage of enucleation has been reduced from 20-23% to 3-6%.[75] A combination of intraocular antibiotic with vitrectomy is effective in the management of posttraumatic endophthalmitis76,77.

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