

## ORIGINAL PAPERS

# PRIMARY BLAST INJURIES TO THE EYE: A REVIEW OF THE EVIDENCE

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## Abstract

**Objectives:** Eye injuries are relatively common following detonations. The majority of these are caused by secondary fragments. Primary blast injury to the eye (from the air-blast alone) has been described historically, but the differentiation of air-blast and fragment aetiologies in the reports is not clear. The aim of this review is to consider the evidence in the literature for true primary blast injury to the eye.

**Methods:** A literature review using Medline, Pub-med and Web of Knowledge databases.

**Conclusions:** Overall, there is little conclusive evidence that primary ocular blast injury occurs in survivors of explosions. However, some case reports do surmise its occurrence and it cannot be unequivocally ruled out. If it does occur, it is more likely to be at high overpressures that will produce life-threatening injuries. The development of enhanced blast weapons may result in an increased incidence. At present however, the most severe and damaging threat to the eyes from blast remains the impact of fragments and debris.

## Introduction

The incidence of eye injuries sustained during combat has steadily increased over the past 150 years. Ocular injuries accounted for 0.65% of all injuries in the Crimea, about 2% in World War Two and had increased to about 9% in Vietnam(1). In one US Combat Support Hospital Emergency department during Operations Desert Shield and Desert Storm, 14% of cases seen were related to eye injuries(2). Over an eight month period in Iraq in 2004, one US Combat Support Hospital saw 207 patients with severe ocular or ocular adnexal injuries(3) (2794 patients were admitted during same time period). Between March 2003 and December 2004, 22.5% of cases evacuated by the US Army from Iraq and Afghanistan were due to eye disease (of which 258/368 were due to battle injuries)(1).

Military surgeons are now trained to expect eye injuries in approximately 10% of battle casualties(4), despite the eyes and adnexae comprising only 0.27% of the erect frontal silhouette. This increase has been attributed to the increased use and efficacy of fragmenting munitions (e.g. land mines) and Armoured Fighting Vehicle warfare (where the externally exposed body area is limited to the head and neck)(5,6). It also reflects increased survival rates from injuries that historically would have been fatal, due largely to the increased incidence of body armour use, and developments in military medicine(6). Fragments (secondary blast injury) are attributed with causing about 80% of military ocular trauma(6,7), but there are uncertainties about the frequency and significance of primary blast injury to the eye, i.e. injuries arising from the blast overpressure.

Blast injuries to the eyes are not purely a military problem.

Munitions such as landmines also affect civilians. Muzaffer et al published a series of 84 non-combatants who suffered eye injuries from land mines(8). Recent terrorist bombings have also brought in to focus blast injuries in the civilian setting; after the Oklahoma bombing, 8% of injured survivors had ocular injuries(9).

There is potential in the future for primary blast injuries to increase in frequency with new weapons systems which are designed to increase the primary blast effects on personnel(10).

This paper aims to review the evidence for primary blast injury to the eye.

## Methods

A comprehensive search of the literature was carried out, using Medline and Pre-medline 1966 to November 2006, PubMed and Web of Knowledge. Search terms included "blast eye injur\*" and "ocular blast injur\*". The bibliographies of relevant papers were examined and cross-referenced.

## Blast injuries

The blast wave arising from an explosion or other high pressure release of gas applies a force to the body which will lead to displacement of the body surface. The acceleration of the body surface also generates an internal stress (pressure) wave. This stress wave interacts with internal interfaces of different densities (for example the capillary walls separating air and blood in the lungs) in a complex manner, creating pressure differentials that can be extremely destructive. Primary blast injury is well documented to affect the lungs, gut and ears (11).

Secondary blast injury refers to damage sustained when objects, accelerated by the initial detonation or subsequent overpressure loads, strike the body – these may be fragments of the explosive casing (primary fragments), or parts of the surrounding environment (e.g. glass, bricks, soil - secondary fragments). Tertiary blast injuries result from the displacement

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of the body (or parts of the body such as limbs) by the blast wave and can lead to avulsion or impact with solid objects.

## Ocular injuries related to blast

Ocular injury related to blast is frequently due to its secondary blast effects – fragments from the weapon and debris. Fragmentation may contuse the eye or adnexa without penetration, or foreign bodies may penetrate and rupture the globe. Particles of sand, glass or dirt accelerated by the blast wave may pepper the surface of the eye. To a much lesser degree, injury to the eye can occur through tertiary blast effects – the orbital region striking a stationary object as the individual is propelled by the blast wave. The existence of secondary and tertiary ocular blast injury is indisputable(3,5,9,12,13). Less conclusive is the evidence presented regarding primary blast injury to the eye.

## Historical reports

Comments indicating primary ocular blast injury are evident in papers dating back to World War Two. Several authors wrote about what may be construed as primary blast injuries to the eye but at this stage terms such as concussive eye injuries are used. Goulden claimed an increase in concussive ocular injury attributable to blast due to the increased efficacy of explosives: from 6% of all ophthalmic casualties in WWI to as many as 40% in WWII – although it should be noted that the author's differentiation between primary and secondary blast injury is indistinct(14). Campbell claimed *'the sudden compression and expansion of the atmosphere caused by blast'* to be one of the three main causes of ocular injury during air raids, noting 'peculiar' effects on the eye that include proptosis due to retrobulbar haemorrhage, iridodialysis, tearing of retinal vessels, choroid rupture, acute iritis and secondary glaucoma(15). Wiener related cases of exophthalmos, choroid rupture and aneurysm of the lateral sinus, without obvious external cuts or bruising, upon which he blames blast exposure(16). Bellows cited thirteen cases of pure blast injury with 'no apparent injuries by impact or penetration of a foreign body', displaying pathologies including optic nerve atrophy, choroidal and retinal lesions, intraocular haemorrhage or vitreous opacity, lens, iris and pupil changes, and extra-ocular muscle paralysis(5).

However, without description of the circumstances of injury and complete reports of the extent and nature of damage sustained, the value that can be attached to these early case studies is limited. Often lacking is even a rudimentary confirmation that no obvious secondary blast injuries were present. Several authors raised the point that where there is suspected primary blast injury, there is often evidence of secondary blast injury also (5,17). If a penetrating injury or intraocular foreign body is present, it is impossible to determine which ocular pathologies have been caused by specific blast effects. Even if it is believed that penetrating injuries are absent, the possibility of non-penetrating contusion by a blunt object can seldom be ruled out. Chiffelle reported very slight pitting of the conjunctival and corneal surfaces, without penetration or 'tattooing' (when debris particles are embedded in the surface of the eye), caused by the impact of flying dust particles upon the eyes of experimental animals subjected to severe blast (17).

The assumption that ocular primary blast injury exists clearly gained authority over time, despite a lack of conclusive supporting evidence. The presence of any number of an extensive, yet undefined range of pathologies is immediately attributed to *'the blast effects of modern high explosives'* (18). Jamra et al present three cases of injury caused by letter bomb explosions (19). The 'bizarre' ocular injuries (including globe rupture, iridodialysis, hyphaema, lens rupture, traumatic cataract, retinal haemorrhage, oedema and macular cyst) of these patients are attributed solely to *'a sudden increase in atmospheric pressure due to the explosion'*. Yet each

casualty suffered lacerations of the adnexae, face and neck, in addition to tears of the conjunctiva and sclera, while one case even describes the peppering of the corneas with explosive – the presence of secondary blast injury is indisputable. Mandelcorn and Hill cite *'a sudden increase in atmospheric pressure due to a blast'* as the cause of orbital blow-out fracture with herniation of the globe into the maxillary antrum. However, as the empty orbit was 'packed with dirt', displacement of the globe by the effects of secondary blast is equally as plausible an explanation (20). Quere et al believe that *'direct contusion of the eye'* by blast was one of the major mechanisms of ocular injury in 132 observed cases, yet undermine their conclusions by describing small skin gaps concealing *'enormous cavities packed with debris'*, and peppering of the face by dirt – which confirm the presence of secondary blast injury (12). Moore et al claim that *'the concussive effect [of car battery explosions] can cause injuries such as hyphaema, angle recession, and retinal oedema'*, without proposing that the acid burns and penetrating injuries also suffered could be responsible (21).

More recently, Zerihun claimed that *'the explosive force that causes concussion of the eye ball'* is responsible for lens dislocation and cataract (22). This is in reference to a paper by Rones and Wilder, who observed 23 cases of such pathology among 104 non-perforating injuries (not necessarily blast-related) to the eye. Yet the case studies in the latter paper suggest that injury was caused by a direct blunt trauma, while the discussion stated that the direction of lens dislocation was dependent 'upon the direction of the blow' (23).

## A mechanism for ocular primary blast injury

Duke-Elder (24) is the first author to offer a view on the mechanism of ocular injury by air blast. Both the positive high pressure shock front and the subsequent wave of lower sub-atmospheric pressure are believed to contribute to injury – according to the author, the latter can be violent enough to result in *'gross disorganisation and even complete avulsion of the globe'*. As the blast wave strikes the eye from an antero-posterior orientation, the pressure wave is transmitted through the incompressible fluid contents of the globe. The cornea is distorted, forcing the aqueous, lens and iris backwards, and driving the vitreous against the posterior pole. The pressure is also transmitted from the centre of the globe outwards. There is forcible expansion of the eye around the equator perpendicular to the direction of impact, causing compensatory circumferential lateral distension, with the effect of the ocular contents being driven against the orbital walls. There is also the confounding damaging effect of the *'establishment of sharp pressure gradients'* between ocular structures that differ in *'inertia, compressibility, cohesion and fixation'*.

Among the many pathologies that he attributes to blast, Duke-Elder described characteristic lesions of the posterior pole that include retinal oedema, choroid rupture and degeneration, and macular lesions; as well as diverse anterior segment pathology, such as subconjunctival and intra-ocular haemorrhage, traumatic iritis, secondary glaucoma, and traumatic cataract. It is a combination of three mechanisms that Duke-Elder believed causes this ocular damage following the impact of a blast wave. These three factors – disruption of cellular function, vascular disturbance, and mechanical tearing of tissue – combine to produce such a varied spectrum of injury following exposure to blast (24).

## Does ocular primary blast injury occur only at high overpressures?

Some authors believe that ocular primary blast injury may indeed occur, but is rarely witnessed as an independent entity.

Galbraith (unpublished MOD report) believed that as *'the liquid-containing eye lies in an orbit filled with fat and surrounded by bony walls'*, it is susceptible to stress and shear waves, as well as distortion in the manner of blunt trauma. However, he suggests that even if such primary blast injury is common, it may be hidden by only occurring at a pressure threshold high enough to result in fatality from other injuries – and where secondary blast injury is also likely to be sustained.

Experimental study has supported this view. Chiffelle discussed research carried out by Richmond et al in the 1960's(17). They demonstrated orbital 'blow-out' fractures in dogs exposed to blast. 'Blow-out' fractures are commonly seen following orbital impact by a solid object such as a ball or fist, which results in a sudden increase in intraocular pressure that fractures the bones of the orbital floor, usually along the line of thin bone that covers the intraorbital canal. Less commonly, fractures involve the medial wall of the orbit, causing portions of the frontal, sphenoid and lacrimal bones that form the lamina papyracea (a thin plate of bone covering the ethmoid sinuses) to project medially into the nasal fossa. It is this latter type of fracture that has been observed experimentally at high blast overpressures (above 140psi [980kPa]), provided that the time to maximal pressure was less than 30ms. (It should be noted that the original Richmond study was carried out using a shock-tube and in their series only eleven fractures were seen in nine of 115 animals exposed. The shock-tube acted to reflect the shock-wave and caused a considerable reflected composite effect; it was the time to the maximum composite pressure being reached that was measured).

After impact of the blast wave, the intra-ocular pressure is elevated and transmitted to the bony walls of the orbit. The pressure wave is also transmitted through the nasal fossae and sinuses, delayed by the small size of the openings into the air spaces. There is, therefore, a critical momentary pressure differential on either side of the orbital bone. Chiffelle believed the mechanism of injury from blast to be similar to injury caused by a solid object: *'the transmission of the pressure pulse by way of the fluid content of the orbit, [is] sufficient to overcome the thin bony plate'* of the lamina papyracea(17). Clinical consequences are documented to include periocular ecchymosis and oedema, enophthalmos, infraorbital nerve anaesthesia, diplopia, nasal bleeding, periocular subcutaneous emphysema, and ocular damage including subconjunctival haemorrhage, hyphaema, angle recession and retinal dialysis. It was also noted that should fracture lines extend posteriorly, as occurred in one animal, a thoroughfare to the meninges will be opened, exposing them to infection.

There appear to be few reports of such fractures in case reports of human blast exposure. Osborn and Mandelcorn noted the presence of orbital blow-out (20,25). Agir et al present a case of a woman injured in a Turkish bombing in 2003 who presented three weeks afterwards with bilateral orbital blow-out fractures and total visual loss(26). The authors felt this was due to the direct blast effect but there was also considerable evidence of secondary blast injuries. Mines et al report six orbital fractures following the Oklahoma bombings, but it is unclear as to what they feel was the cause of these injuries(9). In at least half of these patients, there is evidence of secondary blast injury. Phillips postulated the low human incidence may be due to the differing skull shapes between the two species(27).

Richmond et al (quoted in Chiffelle(17)) also suggested theories for the 'apparently' low number of eye lesions recorded in human blast casualties, when experimental evidence showed their existence in animals. Richmond felt that the high pressure (above 140psi) blast waves which in his study caused eye damage would be likely to be fatal in humans as lung damage

would be caused prior to damage to the eye. As blast injury characteristically causes early mortality, so few patients exposed to blast loads sufficient to cause eye injury would survive to receive medical assistance – particularly in a military setting where considerable delay between injury and medical facilities is common. The presence of severe or fatal injuries in humans may result in orbital blow-out being overlooked clinically or at post-mortem – especially as it is difficult to detect unless associated retrobulbar haemorrhage has resulted in proptosis (27). It was worth noting though that Chiffelle himself wrote that he had *'never observed any significant injury to the ocular globe'* in any animals in his own laboratory or field studies apart from *'slight pitting of the conjunctival and corneal surfaces from flying dust particles'*(17).

Studies undertaken by Zuckerman have also indicated that only relatively high pressures (equivalent to being in 'close vicinity' to a blast) consistently cause retrobulbar and occasional associated conjunctival haemorrhage. No other pathologies (including orbital blow-out) were noted by the author, who believed that other types of injury can be attributed to pre-existing pathological conditions – to accept this theory, the injuries observed in the cases cited above must be deemed to represent a high degree of secondary injury (28). In concordance with Zuckerman, while reporting six cases of eye injury by compressed air jets (see below), Hosni described two cases of retinal dialysis and detachment where the other (uninjured) eye showed peripheral retinal degeneration with vitreoretinal pathologic findings (29). Like Zuckerman, the author considered that the injured eye can be presumed to have been suffering the same changes, so predisposing it to injury.

### High-pressure compressed-air injury to the eye

Indication that ocular injury may result from close proximity to explosions, or high blast pressures from other sources is provided by a number of reports of civilian accidents involving compressed air hoses, such as those used on vehicles tyres. In each case, loss of control of the hose led to the jet of air (compressed to 50-175psi) being directed towards the eyeball.

Despite the pressure being created being similar to that seen in an explosion, it is important to consider what is actually happening, as it is a very different mechanism from that in a blast. The air in a hose is 'flowing' from one area of high pressure to an area of lower pressure, rather than being 'pushed' by an explosion. Therefore the rate at which the compressed air reaches peak pressure is much slower and the pattern of pressure changes observed is different. They are therefore not directly comparable and indeed it may be the jet of gas leads to injuries which are equivalent to the sudden distortion produced in a contusion injury.

The flow from an air hose is also much more localised than that from the detonation of explosive. Overpressures similar to those emitted from the air hoses described in the literature would be expected to cause some degree of injury to the lungs if applied over a large area of the body, and so represent somewhere between a relatively high and very high blast load. However, these cases warrant consideration, as ocular blast injury would undoubtedly be (in part) the result of concussive injury; if blunt trauma can distort the eye and damage any part of it, why should not an explosive wave striking the front of the eye also cause rapid distortion?

Injuries observed in compressed air trauma included reduced visual acuity (in some cases to the extent of no perception of light), orbital emphysema, corneal oedema, increased intraocular pressure, conjunctival and subconjunctival haemorrhage, retinal dialysis and detachment, and optic nerve atrophy(29-38). Conjunctival lacerations were routinely

observed, on occasion caused by direct trauma from the hose(36). More often, the nozzle did not penetrate the eye, and such (micro) lacerations were thought to be caused by the air jet itself. In only two cases (30,38) was there a complete recovery, including visual acuity returning to baseline.

The injuries observed in these cases are to some degree equivalent to the damage alleged to be caused by primary blast – indeed, Hitchings and McGill note that any high-pressure injury can potentially cause injury through contusion or displacement(30). However, it is important to bear in mind that the compressed air injuries were the result of pressures so high that they would only be expected in large blasts or at very close proximity. This supports the arguments proposed by Zuckerman – these compressed-air pressures, if translated into whole body blast overpressures, would be likely to cause severe injury or death, masking any ocular injuries(28), and also increasing the likelihood of concurrent secondary blast injury.

### A confounding case report?

From the evidence presented, the existence of primary blast injury to the eye in the survivors of explosions is inconclusive; but it is also difficult to rule it out completely. Beiran and Miller(39) certainly confound the issue with a case report of a 20 year-old who was exposed to a blast of 500kg explosive at a range of 90m. The individual did not lose consciousness at any point, and both he and three other persons stood nearby could testify that no flying objects struck him. Three bystanders suffered eardrum rupture. The patient suffered mild blurring in the left eye, with conjunctival and ciliary injection, hyphaema and some irido-corneal angle recession, which recovered completely by 3 months. No intra-ocular foreign body or evidence of blunt trauma was evident.

This case appears to suggest that pure ocular primary blast injury occurred, without secondary blast effects, and at a relatively low peak overpressure; an event that supports of the work of Williams and Petras(40;41), and contrasts with the comments of Zuckerman(28). This is perhaps the most reliable source of evidence indicating the existence of ocular primary blast injury.

### Air emboli: an alternative mechanism for ocular injury following blast

Both Chiffelle and Sharpnack et al present an alternative theory to pressure changes and concussive effects, with regard to the mechanisms of ocular injury following blast (17,42). The authors claim that the eye is 'extraordinarily resistant' to blast, being of relatively equal density throughout – which conveys resistance to creation of local stress and shear pressures as the blast wave couples through the body tissue. Instead, cases of transient blindness, hyphaema, and conjunctival haemorrhage following blast may be the result of air emboli within retinal vessels – which are a consequence of alveolar-pulmonary venous fistulae formation in primary blast injury to the lung, and have been demonstrated ophthalmoscopically(27). The authors claim that other ocular injuries can usually be attributed to secondary blast effects, the presence of which are 'difficult to exclude... with absolute certainty'(17).

### Experimental observation of embolic damage to the ocular system

Petras et al investigated the effect of blast on the central visual pathways of the brain, producing conclusions that may explain some of the changes in visual acuity seen in earlier case reports(41). A compressed air-driven shock tube was used on rats to simulate the impact of a pure blast wave, without confounding factors such as fragmentation. The rats were exposed laterally to three levels of pressure blast and recovered

for 7-14 days to maximise the development of pathology.

At post-mortem examination, none of the rats exposed to the lower level had visual system changes, but axonal degeneration was observed in 25% of the middle pressure group and 83% of the high pressure group. Ipsilateral optic nerve degeneration could be tracked to the optic chiasm, and from here, degeneration in the ipsi- and contralateral optic tracts was traced to the midbrain and caudal diencephalic nuclei. The visual functions and interactions of the areas affected (including the lateral geniculate bodies, superior colliculi, and pretectal region) were unclear, and Petras suggested that 'further experimental study is needed to ascertain if blast exposed survivors do in some instances sustain combined injury [to the brain in this manner]... and if this can be correlated with ocular motor deficits'(41). It should be noted that the orientation to blast and the lateral placement of the rats' eyes in the skull resulted in unilateral injury. The frontally-directed placement of human eyes may mean that humans are more susceptible to bilateral injuries, which Petras believes may include loss or impairment of ocular movements, and of the pupillary and accommodation reflexes, as well as retinal haemorrhages, scotomas, retinal detachment and blindness.

No concise theory is presented regarding the mechanism of injury, although offered is the possibility that traumatic cellular injury is caused by the physical compression of the eyeball by the blast wave(24). This study demonstrated permanent injury to the neuro-ophthalmic pathways (retinofugal axonopathy), and at relatively low overpressures – clearly in opposition to Zuckerman who believed that very high pressures are required(28). Indeed, it appears to support Williams, who argued that 'even low pressures suddenly applied to the body can cause injury' (40).

Petras(41) has illustrated damage to the visual system following blast, but this may not be comprehensive evidence that primary blast effects can cause injury to the eye. Damage caused by traumatic compression of the eyeball may have propagated along optic nervous pathways by the mechanisms discussed by Duke-Elder (see above)(24), which could explain the reduction in visual acuity observed in previously cited cases. However, the commonly-held belief remains that pure ocular primary blast only occurs at overpressures much higher than utilised in this experiment (at least 350kPa, or 50psi (28)) – making concussion from air blast an unlikely (but not impossible) cause.

An alternative mechanism is suggested. As the Petras(41) study submitted the entire bodies of the experimental animals to blast, it is possible that the resulting neuro-ocular pathology was due to ischaemic damage caused by air emboli – a known consequence of primary blast injury of the lung(17). The pressures utilised support this theory, as the threshold for lung injury occurs at much lower pressures than that at which ocular injury is believed to occur. This injury mechanism would therefore offer a plausible rationalisation for instances of reduced visual acuity observed in many of the case reports previously discussed. The consequence of this hypothesis is that such disturbances to the visual and neurological systems must therefore be considered the result of lung damage – which would negate the theory of ocular primary blast injury at these pressures.

### Dismissing the air emboli theory? Absence of pathology following blast exposure

It is hardly unexpected that contradictory evidence appears to be presented in another study, carried out by Clemedson et al(43). Rabbits were subjected to one of three levels of blast pressures. In the first group, no pathological changes were

witnessed in the brains of the animals. Above 1420kPa, the authors observed some areas of haemorrhage – believed to be due to tearing of vessels by the shock wave, and vascular permeability (demonstrated by trypan blue dye) – believed to be caused by inflammatory mediator release following air emboli-induced anoxia.

The majority of pathological changes observed were in the cerebellum and diencephalon, with none in the optic nerves or tracts, and were considered ‘rather limited’ – certainly not of the same magnitude as observed in the Petras study, despite the use of much larger overpressures. However, the authors do note that the animals were euthanised and the brains washed out with saline and formalin ten minutes after blast exposure (rather than the 7-14 days of the Petras study), making it ‘possible or even probable that all the changes induced by the blast had not had time to become visible’ (43). Hence, without extension of this study, it is difficult to equate the results with that of the Petras study, and therefore their use is limited in such a role.

## Conclusions

Although there is limited evidence to suggest the existence of unmixed ocular primary blast injury, experimental studies and certain case reports make it impossible to rule out as a contributory cause of pathology, particularly if secondary blast effects are also believed to play a part.

If ocular primary blast injury does exist, the development of enhanced blast weapons may result in an increased incidence. At present however, the most severe and damaging threat to the eyes from blast remains the secondary effects of fragmentation and particle acceleration.

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