Anatomical Manifestations of Primary Blast Ocular Trauma Observed in a Postmortem Porcine Model

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Purpose. We qualitatively describe the anatomic features of primary blast ocular injury observed using a postmortem porcine eye model. Porcine eyes were exposed to various levels of blast energy to determine the optimal conditions for future testing.

Methods. We studied 53 enucleated porcine eyes: 13 controls and 40 exposed to a range of primary blast energy levels. Eyes were preassessed with B-scan and ultrasound biomicroscopy (UBM) ultrasonography, photographed, mounted in gelatin within acrylic orbits, and monitored with high-speed videography during blast tube impulse exposure. Postimpact photography, ultrasonography, and histopathology were performed, and ocular damage was assessed.

Results. Evidence for primary blast injury was obtained. While some of the same damage was observed in the control eyes, the incidence and severity of this damage in exposed eyes increased with impulse and peak pressure, suggesting that primary blast exacerbated these injuries. Common findings included angle recession, internal scleral delamination, cyclodialysis, peripheral chorioretinal detachments, and radial peripapillary retinal detachments. No full-thickness openings of the eyewall were observed in any of the eyes tested. Scleral damage demonstrated the strongest associative tendency for increasing likelihood of injury with increased overpressure.

Conclusions. These data provide evidence that primary blast alone (in the absence of particle impact) can produce clinically relevant ocular damage in a postmortem model. The blast parameters derived from this study are being used currently in an in vivo model. We also propose a new Cumulative Injury Score indicating the clinical relevance of observed injuries.

Keywords: trauma, blast impact, ultrabiomicroscopy (UBM), primary blast injury, histopathology

Blast injuries generally are categorized according to the interaction of the body with the shock wave (primary), energized particulate matter (secondary), blast wind (tertiary, includes bodily displacement and structural effects), and other effects (quaternary).[1] Although case studies have long implicated primary blast in ocular trauma,[2,3] very few experimental data have been collected to document this phenomenon. Petras et al.[4] used a shock tube to expose rats to primary blast, and characterized resulting injuries to the optic nerve and brain. Our own prior research has focused upon mechanisms and effects of injury caused by particle impact.[5–7] Previous studies, although inconclusive, have not ruled out that primary blast may create clinically relevant ocular damage. The mechanisms of primary blast induced injury likely differ from those caused by other types of blast related injuries (secondary, tertiary, and quaternary). Primary blast mechanically loads the exposed eye surface, orbital rim, and periorbita fairly uniformly relative to loading produced by secondary and tertiary blast. These latter categories of ocular injury have been reasonably well studied, but primary blast has been relatively neglected in the literature. This may be due to the paucity of reports of isolated ocular injuries attributable solely to primary blast, as well as the technical challenges associated with the experimental study of pure primary blast effects.

Cockerham et al.[8] noted the importance of closed globe injuries in soldiers injured by blast. Similar to our previous studies of blunt impact without penetrating injuries, we hypothesize that distributive loading will tend to induce closed globe injuries. In those studies, empirical ballistic imaging and computational modeling confirm such injury was associated with globe distortion and postimpact pressure oscillations. We tested this hypothesis by exposing porcine eyes to blast with a shock tube. The use of a shock tube allows for the production of a controlled primary blast component without secondary or tertiary effects. Thus, the injuries

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observed are most likely from primary blast, specimen preparation, or postmortem degradation.

In a recent 60 year review of 244 articles reporting injuries associated with explosive blasts, ocular injuries occurred at very high frequency, afflicting more than 28% of blast survivors.[9] Ocular injuries now account for 13% of all battlefield injuries and are the fourth most common military deployment related injury.[10] The incidence of ocular injury in combat is 20 to 50 times greater than one would expect based on exposed surface area alone.[11] Eye injury rates in active duty military personnel increased from 1996 to 2005, reaching a maximum of 26 and 21 per 1000 person years in men and women, respectively.[12] Approximately 80% of ocular injuries in military conflicts are associated with blast fragmentation.[13,14] One potential explanation for the high incidence of ocular injury relative to its exposed area is that the eye may be more mechanically susceptible to blast related injuries than other tissues. In addition, soldiers nearly always wear head and chest protection, but not eye protection. In the majority of these ocular injury cases (85% 90%), the subjects were not wearing eye protection.[14,15]

Shock tubes have been used since the 1960s to simulate the effects of blast on small and large mammals[16] as well as military vehicles and equipment.[17] Shock tubes reliably produce overpressure representative of the Friedlander waveform, which is an idealized description of a free field explosive charge.[18] The shock tube used in the present study was designed to produce shock waves similar to low end pressures generated by improvised explosive devices (IEDs).[19] Shock tubes have been instrumental in producing the Bowen Curves, which correlate a given pressure and duration with a probability of lung damage and survival probability.[20] To date, analogous curves have not been generated for ocular trauma due to blast to our knowledge.

The paucity of documented isolated primary blast injuries emphasizes the importance of the present study, an investiga tion to generate qualitative anatomical descriptions of ocular damage arising from primary blast exposure in a porcine model. We are particularly interested in understanding injuries that are not catastrophic (i.e., closed globe). Therefore, we examined the literature and selected an experimental pres sure duration space based on the lower end reported for IEDs in the literature.[19] We speculated that if ocular injuries can be caused by overpressure, the severity and likelihood of ocular trauma will increase with the energy content of the primary blast as observed in previous experiments with blunt impacts of paintballs with the eye.[5,7]

Abbotts et al.[3] performed a thorough literature review of blast induced eye related injuries. While thousands of docu mented ocular injuries have been caused by blast trauma, almost all integrate primary, secondary, and tertiary causes. Although anecdotal, a handful of case reports subsequent to their review have documented several isolated ocular injuries attributed by their investigators to only primary blast effects. These included blowout fractures,[21–23] vitreous hemorrhage,[24] hyphema and iridocorneal angle recession,[2] and retinal detachment,[24,25] an injury commonly associated with primary blast. However, in most cases reported in the literature, secondary and tertiary effects cannot be entirely ruled out.

Methods

Sample Preparation

Porcine eyes, including eyelids and extraocular muscles, were purchased from Animal Technologies, Inc. (Tyler, TX) and shipped overnight on wet ice. All animal tissues were handled in accordance with the ARVO Statement for the Use of Animals in Ophthalmic and Visual Research, and protocols approved at each institution. The superior sclera first was marked using a surgical marker based on eyelid position to allow repeatable identification of locations on each eye. Skin, muscles, eyelids, and fatty tissues then were removed to expose the surface of the globe, placed in Hanks balanced salt solution (HBSS; Fisher Scientific, Hampton, NH), and transported to the pathology laboratory.

Despite transfer on ice, due to the hydraulic conductivity of the scleral membrane and the necrotic state of the tissue, the IOP of the porcine eyes initially was low, evidenced by a flaccid cornea, soft eyewall, and failed readings from our Tono Pen VET (Dan Scott & Associates, Westerville, OH). It often was necessary to reinflate the porcine eyes with HBSS so that they were at a normal pressure level (10 20 mm Hg) before scanning and again before overpressure treatment. We found that injections at the pars plana increased the fluid content of the posterior segment, allowing subsequent anterior chamber injections to maintain a normal IOP. At the pathology laboratory, each eye was reinflated via paracentesis of HBSS using a 30 gauge needle until the IOP was between 10 and 20 mm Hg as determined using the Tono Pen. B scan (Compact Touch; Quantel Medical, Bozeman, MT) and ultrasound biomicroscopy (UBM, OIS 100; i Science Interventional, Menlo Park, CA) then were used to assess the condition of each pressurized eye before blast exposure. B scans were taken from 3 to 9 o'clock, 6 to 12 o'clock, and 9 to 3 o'clock axially and posterior near the optic nerve to visualize the anterior chamber. The UBM was used to image each eye from pars plana, equator, and peripapillary regions from clock hours 12, 3, 6, and 9. Eyes were rejected from the study if preexposure pathology was observed during this prescreening process. After screening, eyes were stored refrigerated (4°C) overnight in HBSS, then transported to the shock tube laboratory.

Several acrylic orbits were fabricated with internal dimen sions and geometry similar to the orbital structure.[5,20,26] A plastic cup with spherical bottom (diameter roughly equivalent to the porcine globe) was placed in the center of the acrylic orbit and the remaining internal volume filled with a liquid gelatin mixture (Knox Gelatin; Kraft Foods, New York, NY), which was cooled overnight before delivery of the eyes (Fig. 1a). This gelatin has been shown to provide nearly equivalent stiffness as the extraocular muscles and associated periorbita.[27] The orbit mimic used in these trials was created by using a small plastic cup with comparable curvature and depth as the porcine eye to make a negative mold in the gelatin while in its liquid phase. This gelatin mold then was cooled to 4°C for one half hour until firm. It was necessary to adjust the depth of the porcine eye by cutting/rearranging the solid gelatin posteriorly because of differing eye sizes, and to accommodate variations in optic nerve lengths to ensure that the limbus and cornea would remain outside the gelatin in a neutral gaze attitude. Each eye was reinflated via pars plana injection with HBSS and set in place within the gelatin. Additional liquid gelatin then was added to ensure maximal surface contact between the gelatin of the orbit mimic and porcine eye (Fig. 1b). This added gelatin was cooled via water bath before pouring while still in a liquid state at room temperature. The orbit mimic containing the porcine eye then was placed in the 4°C refrigerator for at least one half hour to obtain maximal rigidity. During refrigeration, each eye was covered with parafilm to prevent dehydration of the specimen.

The above approach was adopted after preliminary studies demonstrated that direct immersion of the globes into warm or hot gelatin induced delamination of the retina, choroid, and sclera. Whether these effects were due to thermal or osmotic effects (gelatin is a hygroscopic ionic polymer) is unclear.

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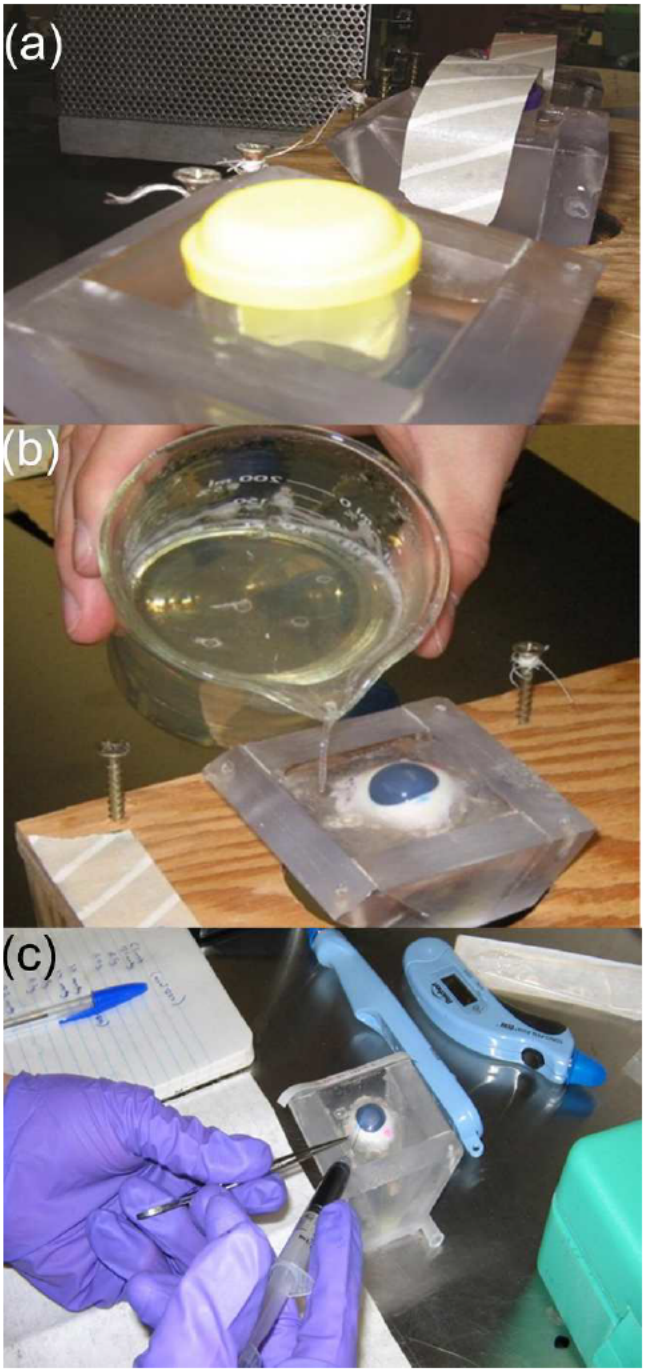

FIGURE 1. Preparation of the eye for blast exposure. (a) Gelatin is preformed within the acrylic holders. (b) Eyes are oriented and set within the preformed gel and sealed in place. (c) Just before blast testing, HBSS is injected in to the anterior chamber. The needle is inserted nearly parallel to the corneal surface in an attempt to provide for some degree of self sealing once the IOP is raised.

Moreover, the two stage temperature controlled preparation method facilitates more faithful and uniform reproduction of the neutral gaze boundary conditions, securing the eye while minimizing resistance to rotational duction.

Just before blast exposure, HBSS was injected into the anterior chamber via a shallow angle through a 30 gauge needle port paracentesis tangential to the limbus in the peripheral cornea to raise the IOP into the estimated high normal range (Fig. 1c). The eye acrylic orbit assembly then was photographed and placed into a rigid mount inside the shock tube. Once secured, the IOP was measured again and recorded. The eye acrylic orbit was tilted 20° laterally to simulate the convergence of the human eye and placed 25 cm into the expansion cone. The test chamber was cleared, the driver section of the shock tube pressurized, and the blast test conducted as detailed below.

For each day of testing, this procedure was repeated for several exposed eyes and one control eye. The control eye was treated in an identical fashion to the test eyes and placed into the expansion cone for several minutes, but was never subjected to an actual overpressure. After the test, IOP was measured and recorded; the eye holder assembly was removed from the rigid mount, and another series of photographs taken. The eye specimen then was removed from the gelatin and placed into an HBSS filled container. The containers then were transported on ice to the pathology laboratory for masked post test damage assessment.

Upon arrival, another corneal HBSS injection through a 30 gauge needle port paracentesis tangential to the limbus was used so that the IOP was between 10 and 20 mm Hg at time of blast exposure. Damage to each eye then was evaluated and documented using a combination of B scan and UBM ultrasound imaging along the meridians and directions examined previously. The masked specimens then were placed in formalin in preparation for detailed examination via manual dissection (in which the anterior surface was removed with a diamond knife) or histologic analysis. Anterior chamber and optic nerve status were assessed further via stained paraffin sections of a subset of specimen eyes representing controls and the full primary blast testing range.

All told, 53 eyes were treated in this way. A total of 13 were used as controls while the balance (40) were exposed to primary blast. Approximately 10% of eyes received from Animal Technologies were excluded because the B scan and UBM ultrasonic prescreening demonstrated the presence of preexisting damage to the eye. Two exposed eyes were excluded from analysis as they were avulsed from the gelatin during exposure.

Additional eyes were used in preliminary testing to develop the methods described above. We found that inadequate inflation of the eye before blasting resulted in much more extensive damage than that observed in eyes with physiolog ical IOP values. It also was determined that immersing control eyes in formalin before the postultrasound examination introduced artifacts in nearly every preliminary test control eye, including detachments that were not present in the preimmersion imaging, scleral shrinkage with radial folds that caused chorioretinal detachment, and obvious change in the tactile characteristics of the globe and coloration changes. For this reason no formalin was used until completing all postimpact UBM and B scan analyses. All eyes subsequently were placed in formalin for preservation and subsequent histopathology that focused upon angle morphology anteriorly, and optic nerve and peripapillary structural changes posteri orly.

Shock Tube

Blast exposure testing at the shock tube lab was accomplished using a 17 inch diameter compressed air driven shock tube (Fig. 2; Applied Research Associates, Rocky Mountain Division, Littleton, CO). The eye specimen and acrylic orbit were placed within the expansion cone of the shock tube and isolated from the driver section by one or more aluminum disks of 0.016 inch thickness and 23.5 inch diameter. Pressurization of the driver section causes the disks to rupture. This rupture generates a Friedlander style pressure wave that travels down

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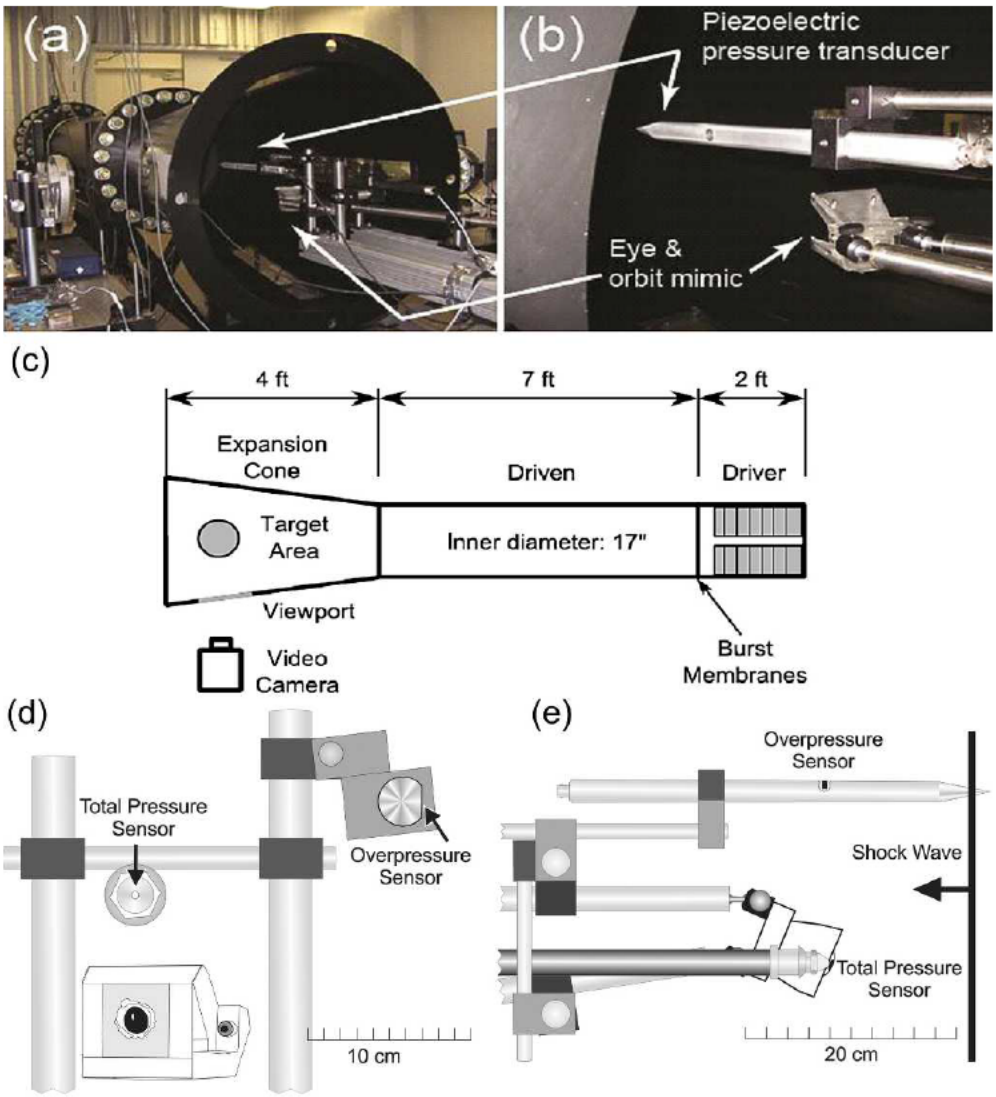

FIGURE 2. (a) Photograph of the 17 inch diameter shock tube used for blast exposure testing. (b) Close up showing arrangement of acrylic orbit and sensor array. (c) Dimensioned schematic of the shock tube. (d) Front and (e) top views detailing the position of sensors around the eye.

the tube toward the eye specimen (Figs. 2a, 2b). The peak pressure of the shock wave is controlled by the number of disks bolted into place between the driver section and the blast tube. Experiments performed using one to six aluminum disks resulted in a range of peak static pressures from approximately 7 to 22 psi (48 152 kPa).

Each blast experiment was documented using high speed videography at 15,000 frames/s using a Fastcam Ultima APX (Photron USA, Inc., San Diego, CA). Static (over) pressure and total pressure were recorded using piezoelectric pressure transducers (respectively, Model 137A23; PCB Piezotronics, Inc., Depew, NY and XTEL 190 7BARA; Kulite Semiconductor Products, Leonia, NJ) as shown in Figures 2d and 2e. Both pressure probes were factory calibrated with appropriate certificates of conformance provided by the manufacturer. Pressure signals were recorded at 200,000 samples per second using a Synergy P Data Acquisition System (Hi Techniques, Inc., Madison, WI). The pulse duration of each experiment was taken as the point where the initially positive phase of the waveform reached zero pressure, (i.e., the negative phase of the waveform is ignored, Fig. 3a). The specific impulse was calculated by time wise integration of the entire waveform.

RESULTS

The shock tube produced pressure waves that closely approximated the expected Friedlander pressure time wave form produced by many explosive devices (Fig. 3a). The peak pressure was not independent of the duration of the positive phase: as the peak pressure increased, the duration increased as well (Figs. 3b d). The peak pressure of the blast increased by approximately 28 kPa (4 psi) per disk ($R^2 = 0.938$). The impulse increased by approximately 0.0338 kPa s (0.0049 psi s) per disk ($R^2 = 0.934$). Table 1 shows the number of eyes exposed to each condition.

High speed video indicated rapid axial oscillation of eyes even at low blast levels. In rare cases at higher blast energies, the eye translated away from the incident blast, rebounded, then completely avulsed from the gelatin. Please note the avulsed eyes were excluded from the analysis.

Using terminology consistent with The Birmingham Eye Trauma Terminology (BETT),[28] the majority of ocular injuries from the experimental blast overpressures were found to be lamellar. Pathology was grouped into three zones, following the convention of Pieramici et al.[29] Incidence of tissue damage

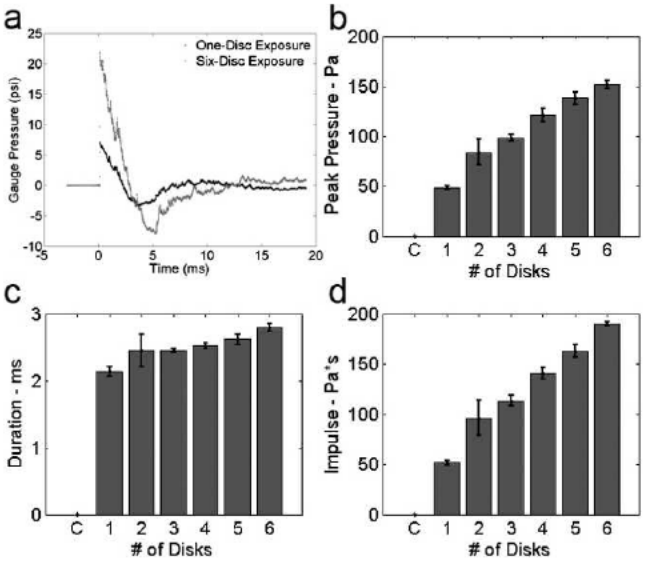

FIGURE 3. (a) Typical pressure time data from two blast exposures demonstrate that the shock tube produces pressure waves that closely mimic the Friedlander waveform produced by explosive devices. (b–d) Histograms showing mean (*error bars* show ±SEM) peak pressure, duration, and impulse for a given shock tube configuration. Pulse durations were taken as the time when the initially positive phase of the wave form reaches zero pressure. Impulse was taken as the integral of the initial positive phase of the pressure data.

was found to increase with peak overpressure in Zone 1 (external surface) and Zone 3 (internal posterior segment), while injuries in Zone 2 (anterior segment) occurred over a wide range of blast energy levels (Table 1). Common findings included angle recession, internal scleral delamination (Fig. 4a) cyclodialysis, peripheral chorioretinal detachments (Figs. 4b, 4c), and radial peripapillary retinal detachments (Fig. 5). No full thickness openings of the eyewall were observed in any of the eyes tested. Corneal epithelial disruption was observed ubiquitously in exposed and control eyes, and, thus, was not included as a Zone 1 injury. Macroscopic injury of the iris occurred infrequently and no obvious examples of pupillary sphincter rupture were observed in these eyes lacking pupillary sphincter tone. Anterior segment uveal damage generally was restricted to the angle, where it was common. Anterior lens capsular damage also was not observed, but zonular dehiscence was noted along with some posterior capsular ectasia in multiple instances. The sclera demonstrated the strongest associative tendency for increasing injury with increased overpressure. There was no obvious impulse threshold value for the appearance of lower grade injury to any ocular structure, but there were such thresholds in evidence for more extreme tissue damage levels readily apparent for the optic nerve (125 Pa s), anterior chamber (100 Pa s), and sclera (90 Pa s). Numerical correlation analyses are beyond the scope of the present descriptive anatomic survey, and will be presented in detail in a subsequent article.

Pathology—Zone 1 (External Surface)

Scleral delamination and multiple chorioretinal detachments were observed before fixation using B scan and UBM, and were confirmed through dissection and histopathology. Figure 4a shows a chorioretinal delamination following exposure to 138 kPa (20 psi) blast. Figure 4b shows a detachment in an eye that was exposed to 113 kPa (16 psi) blast. These lamellar lacerations occurred in multiple meridians. The UBM scans also commonly showed chorioretinal disruption with the probe oriented perpendicular to the equator and with probe directed posteroanteriorly. Histopathology of this eye also showed contra coup peripapillary scleral delamination (Fig. 4c).

Pathology—Zone 2 (Anterior Segment)

Angle obliteration was observed via histopathology compared to the control (Fig. 5a) after the eye was exposed to peak overpressure of 113 kPa (16 psi; Figs. 6b, 6c). The UBM generally was unable to resolve this injury. However, histopa thology revealed the angle as a common location for injury, even for porcine eyes exposed to low peak overpressures.

Pathology—Zone 3 (Internal Posterior Segment)

Images of the optic nerve head (ONH) were taken during manual dissection and from histopathology (Fig. 6). The ONH of the control eye was normal when viewed through the vitreous after removal of the anterior chamber (Fig. 6a). The ONH of an eye exposed to a 207 kPa (30 psi) blast showed retinal elevations coinciding with the location of blood vessels originating from ONH as viewed through the vitreous after removal of the anterior chamber (Fig. 6b). Examples of such injuries may be due to rapid oscillations in the tissue following blast exposure. Such oscillations could induce large strains in regions with density gradients, such as at the blood vessel retina interface.

DISCUSSION

We developed a method for observing closed globe injuries due to primary blast. This method produced a broad array of closed globe injuries that would seriously compromise visual function. This adds to our understanding of blast induced ocular injury by offering an objective assessment of which injuries were most likely due to blast exposure. Preimaging each eye using ultrasound allowed exclusion of eyes that otherwise would contribute artifacts. This improves confi

TABLE 1. Exposure Matrix and Damage Incidence

	Static Overpressure			Total Overpressure			Eyes Having Damage Scores 2 or Higher			
	Impulse	Peak	Duration	Impulse	Peak	Duration				
# Eyes	Pa-s	kPa	ms	Pa-s	kPa	ms	Sclera	Angle	Choroid	Retina
13	0	0	0	0	0	0	1 (8%)	1 (8%)	4 (31%)	4 (31%)
8	26 54	22 40	2.07 2.23	28 61	48 86	2.18 2.28	1 (13%)	2 (25%)	4 (50%)	4 (50%)
11	80 119	70 102	2.26 2.48	100 161	129 175	2.42 2.89	1 (9%)	0 (0%)	2 (18%)	2 (18%)
8	128 148	113 137	2.42 3.08	177 211	191 242	2.93 3.33	3 (38%)	3 (38%)	5 (63%)	3 (38%)
9	158 190	134 155	2.50 2.90	231 292	249 308	3.01 3.33	5 (56%)	1 (11%)	4 (44%)	5 (56%)

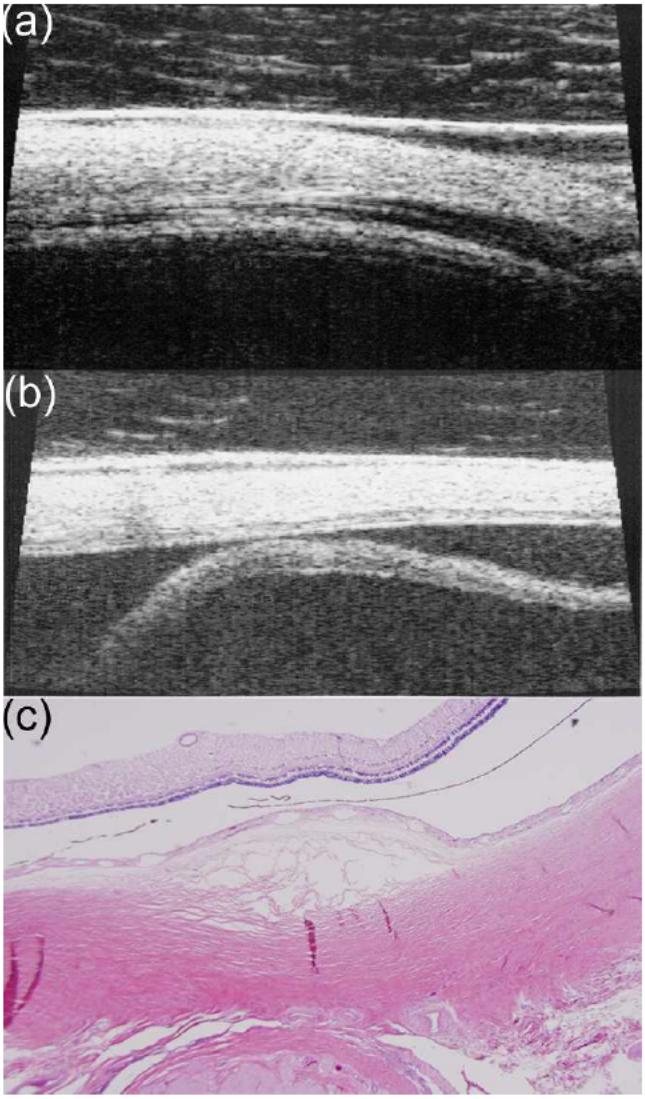

FIGURE 4. Scleral delamination and multiple chorioretinal detachments were observed before fixation using B scan and UBM, and were confirmed through dissection and histopathology. (a) Chorioretinal delamination following exposure to 138 kPa (20 psi) blast. (b) Peripapillary retinal detachment observed using UBM following a 113 kPa (16 psi) blast. (c) Apparent contracoup effect on the sclera and retinal pigment epithelium at the disk margin in the same eye as shown in (b).

dence that any damage to the eye after blast exposure is due to the effects of primary blast rather than preexisting damage.

A notable strength of this study is the evaluation of the blast trauma with the complimentary methods, including UBM, B scan, gross dissection, and histopathology. Specifically, the scans give very clear information about the chorioretina pre and postimpact without the artifacts often produced by tissue dissection. Conversely, the imaging methods were unable to resolve angle recession, while histologic sections gave more reliable information regarding anterior segment damage. In combination, these modalities assist in the primary objective of identifying intraocular injuries that could have lifelong adverse effects.

The increase in severity and occurrence of injury is not a deterministic event. These results support the idea that certain ocular tissues are more sensitive to increases in blast energy than others. The retina appears to be very sensitive to

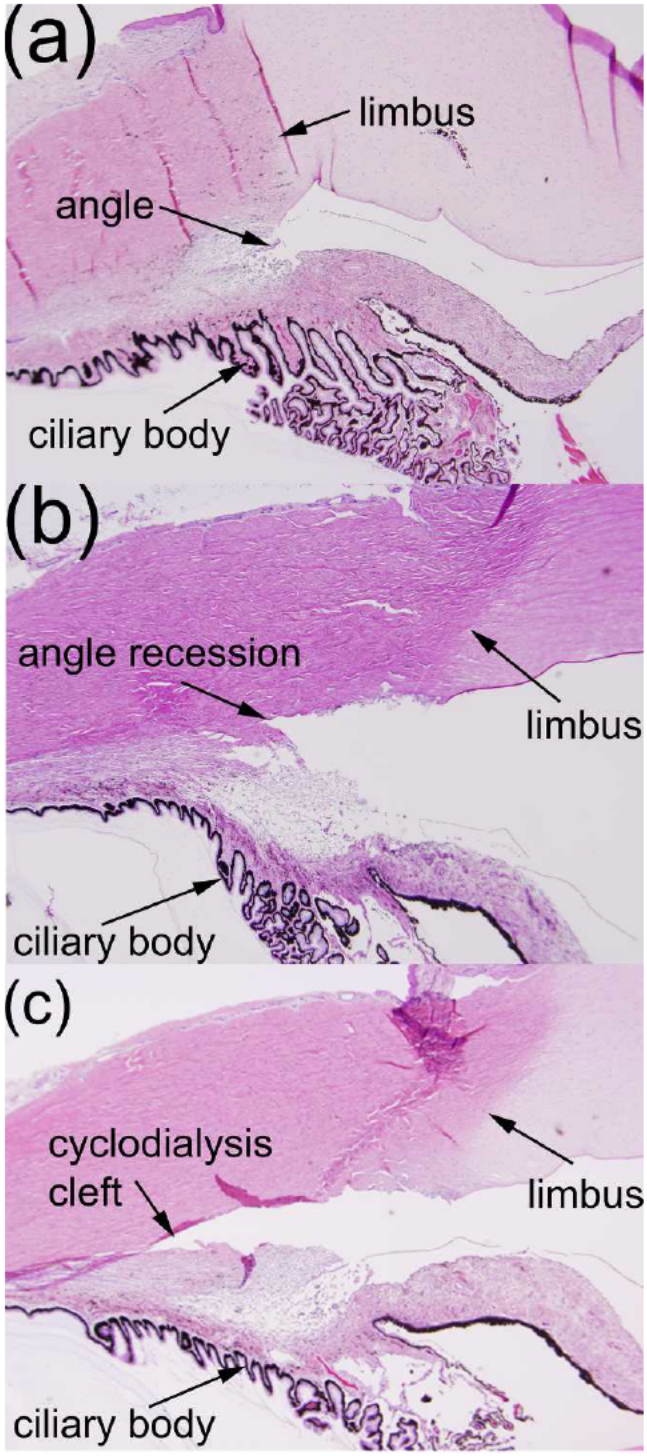

FIGURE 5. Histopathology of the porcine eye angle in (a) a control eye, (b, c) eyes exposed to 113 kPa (16 psi) blasts. Angle recession (b) and cyclodialysis (c) were observed. Damage to the angle was observed commonly even after low peak overpressure exposures.

increasing blast energy. Previous blunt impact studies conducted with paintballs found that ocular damage severity increased with energy.[5–7] The present study found a similar increasing trend in ocular damage with increasing blast energies, though globe distortion and movement were far less than in the paintball study. This may be due to several factors, such as the lower energies associated with the blast,

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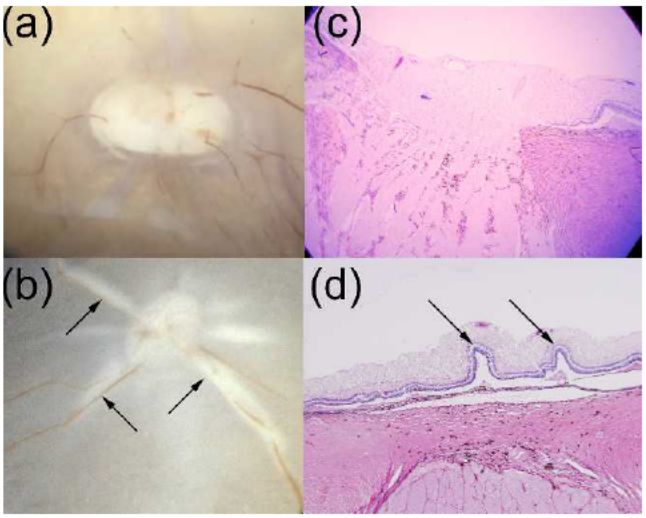

FIGURE 6. Images of the optic nerve head (ONH) taken during manual dissection (*left*) and from histopathology (*right*). (**a**) Control eye showing clear ONH as viewed through the vitreous after removal of the anterior chamber with typical postmortem changes. (**b**) Eye exposed to 207 kPa (30 psi) blast shows retinal elevations coinciding with the location of blood vessels (*arrows*) originating from ONH as viewed through the vitreous after removal of the anterior chamber. (**c**) Control eye section shows ONH and surrounding tissues with typical postmortem changes. (**d**) Eye exposed to 126 kPa (18 psi) blast shows retinal folding in the immediate vicinity of blood vessels (*arrows*). Such injuries may be due to rapid oscillations in the tissue following blast exposure. Such oscillations will induce large strains in the region of density gradients, such as at the blood vessel retina interface.

simultaneous compression of the periorbita, and distribution of the energy over the entire exposed surface rather than concentrating the load at the point of projectile impact. However, we observed a broad array of ocular injuries. Petras et al.[4] reported increased incidence and severity in rats exposed to overpressures of 104 to 173 kPa (15 25 psi), similar to the range of static pressures found in the present study to be associated with a greater incidence of ocular injuries. Hines Beard et al.[30] found an increase in ocular injury and mortality with peak pressure, using a compressed gas pulse with a pulse duration much longer than a survivable blast wave (~100 ms vs. 1 4 ms).

Several finite element models of blast recently have been developed. Rossi et al.[31] modeled the effects of primary blast on the eye suggesting injury potential to the retina, choroid, and optic nerve, based on calculated pressures orders of magnitude higher than published tensile strengths for those materials. They note that this occurred with blast overpressure as low as 75 kPa. Stitzel and Weaver[32] reported risk of hyphema, lens dislocation, and retinal damage. Bhardwaj et al.[33] reported possible risk of injury at muscle attachment points on the sclera, as well as the potential risk of corneal abrasion and hyphema. The present postmortem model cannot demonstrate hyphema or injury at extraocular muscle inser tion. However, retinal detachments were found commonly in eyes exposed to blast.

Porcine eyes are known to have a higher mechanical strength than human eyes.[20] Therefore, the use of porcine eyes in this study should give conservative estimates of injuries that might occur in human eyes due to primary blast. That significant injuries were observed in porcine eyes likely implies primary blast would induce more serious injury in the human eye.

Control eyes experienced some apparent damage that clearly was not due to blast exposure. This may indicate continual degradation of the porcine eyes overnight after the prescreening ultrasound was performed, some damage inflict ed to the eye due to the presence of the gelatin and/or hot lights required for high speed video, or some other artifact of the preparation. Notwithstanding these factors, the incidence and severity of this damage increased with impulse and peak pressure suggesting that primary blast modulated these injuries.

The use of a postmortem model necessarily introduced some "false positive" damage due to tissue degradation and preparation. However, the use of preblast imaging excluded eyes that had significant artifacts before blast exposure. This imaging was useful especially in an ex vivo model as imaging could be performed not only using an anterior approach (the only approach available in vivo), but also equatorial and posterior approaches. This allowed detection of relatively minute damage before and after blast exposure. The ability to detect slight damage also might explain the relatively high incidence of false positives in control samples (8% 31% depending on the tissue), whereas such artifacts would be missed entirely by studies that use less rigorous screening methodologies. In all tissues considered, the incidence of damage was higher in eyes exposed to blast than in controls.

Current ocular trauma scores, like the BETT[28] or the Ocular Trauma Score (OTS)[34] subdivide open globe injuries between lacerations and ruptures, while subdividing closed globe injuries between contusions and lamellar lacerations. Cocker ham et al.[35] noted a need for the development of a universal parlance in describing blast related ocular injuries. Such a scale must account for the full range of ocular injuries extending from the subtle, closed globe nature of primary blast injuries observed here up to and including globe rupture. We have attempted to deliver a detailed anatomic description of closed globe trauma arising due to primary blast in a postmortem model.

The practical application of these new findings will require a meaningful composite scoring algorithm for severity of injury. Scaled algorithms for the severity of the damage to the individual component tissues of the eye and the classic anatomic subsections of the eye have been devised for forthcoming analyses of the association of blast overpressure with the extent of ocular injury. Ultimately, it will be necessary to collate these individual focal injury data into a proposed clinically relevant Composite Injury Scale that addresses the practical needs of those engaged in the treatment of ocular injury or development of protective eyewear (Table 2).

It is apparent from the present study that primary blast overpressure can produce injuries that conform to each of these proposed categories. For example, many of the isolated smaller peripheral choroidal detachments would be Category 1 injuries. Many of the isolated angle recession injuries would lead to chronic glaucoma, requiring filtering or tube shunt surgery (Category 2). The posterior peripapillary stellate retinal detachments would all require timely vitreoretinal surgical intervention to avert blindness, with inevitable

TABLE 2. Proposed Composite Injury Scale

0	The eye is undamaged
1	The eye has some damage, but should heal fully on its own
2	The eye has damage that will require surgery to repair, leaving chronic pathology
3	The eye has damage that might be repairable with surgery, with severe visual loss
4	The eye is likely damaged beyond meaningful functional repair

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functional loss in even the most successful cases (Category 3). Eyes with extensive peripapillary axonal rupture would be expected to develop blinding levels of traumatic optic neuropathy unlikely to respond sufficiently to any current therapy to avert functional blindness in the affected eye (Category 4). When injuries to multiple ocular tissues coexist, the likelihood of therapeutic failure increases: therefore, the integration of the focal and composite injury scales would tend to elevate an eye with multiple Category 2 injuries to Category 3, and any combination of Categories 3 and 2 injuries to Category 4.

This study used a postmortem model of the porcine eye. Repeated repressurization of the eye could have induced artifacts. The decrease in inflation pressure with time is a limitation of the reinflation method. The presence of gelatin may induce osmotic, desiccant, and thermal effects on the eye. Postmortem degradation may make eyes more susceptible to blast damage; in vivo studies will be required to confirm that the living eye is susceptible to primary blast injury.

Continued experimentation in this area will allow more robust correlation between the blast wave characteristics and frequency of trauma response. Ultimately, a probabilistic regression model predicting the likelihood of a specific type of injury similar to the Bowen curves could be combined with data from novel battlefield pressure sensors currently under development (see the report of Cullen et al.[36]) to enable informed treatment by medical staff. A parallel computational study seeks to use dynamic finite element modeling of the blast exposure to understand the mechanisms of these injuries (Gray W, et al. *IOVS* 2013;54:ARVO E Abstract 3045), and others recently have taken a related approach to the problem.[33] Such modeling will serve as a rapid, inexpensive method for testing various protective eyewear solutions that might mitigate blast damage to the eye.

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