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
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.19 Ocular injuries now account for 15% 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 what 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.13,15

Shock tubes have been used since the 1960s to simulate the effects of blast on small and large mammals16 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 investigation 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 protocol. We are particularly interested in understanding injuries due to blast to our knowledge.

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 conjunctiva were removed to expose the cornea 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 eyelid, and failed readings from our Tono-Pen VET (Dan Scott & Associates, Westerville, OH). It was necessary to reinflate the porcine eyes with HBSS to increase 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-scans (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 dimensions 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 extracocular 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.
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 posttest 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 physiologic 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 posteriorly.

**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
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 waveform 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), 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. Incidence of tissue damage
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) cyclodislocation, peripheral chorioretinal detachments (Figs. 4b, 4c), and radial peripapillary retinal detachments (Fig. 5). No full-thickness openings of the eyeball 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 some 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, histopathology 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-

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**Table 1. Exposure Matrix and Damage Incidence**

<table>
<thead>
<tr>
<th># Eyes</th>
<th>Impulse</th>
<th>Peak</th>
<th>Duration</th>
<th>Impulse</th>
<th>Peak</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pa-s</td>
<td>kPa</td>
<td>ms</td>
<td>Pa-s</td>
<td>kPa</td>
<td>ms</td>
</tr>
<tr>
<td>10</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>8</td>
<td>26–54</td>
<td>22–40</td>
<td>2.07–2.23</td>
<td>28–61</td>
<td>48–86</td>
<td>2.18–2.28</td>
</tr>
<tr>
<td>11</td>
<td>80–119</td>
<td>70–102</td>
<td>2.26–2.48</td>
<td>100–161</td>
<td>129–175</td>
<td>2.42–2.89</td>
</tr>
<tr>
<td>9</td>
<td>158–190</td>
<td>134–155</td>
<td>2.50–2.90</td>
<td>231–292</td>
<td>249–308</td>
<td>3.01–3.33</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Eyes Having Damage Scores 2 or Higher</th>
<th>Sclera</th>
<th>Angle</th>
<th>Choroid</th>
<th>Retina</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sclera</td>
<td>1 (8%)</td>
<td>1 (8%)</td>
<td>4 (31%)</td>
<td>4 (31%)</td>
</tr>
<tr>
<td>Angle</td>
<td>1 (13%)</td>
<td>2 (25%)</td>
<td>4 (50%)</td>
<td>4 (50%)</td>
</tr>
<tr>
<td>Choroid</td>
<td>1 (9%)</td>
<td>0 (0%)</td>
<td>2 (18%)</td>
<td>2 (18%)</td>
</tr>
<tr>
<td>Retina</td>
<td>3 (38%)</td>
<td>3 (38%)</td>
<td>5 (63%)</td>
<td>5 (38%)</td>
</tr>
</tbody>
</table>
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 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,
Primary Blast Ocular Trauma

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 inflicted 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% originating on the tissue surface), 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. Cockerham et al.55 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 vitrectomy/surgical intervention to avert blindness, with inevitable

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**Table 2. Proposed Composite Injury Scale**

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>0</td>
<td>The eye is undamaged</td>
</tr>
<tr>
<td>1</td>
<td>The eye has some damage, but should heal fully on its own</td>
</tr>
<tr>
<td>2</td>
<td>The eye has damage that will require surgery to repair, leaving chronic pathology</td>
</tr>
<tr>
<td>3</td>
<td>The eye has damage that might be repairable with surgery, with severe visual loss</td>
</tr>
<tr>
<td>4</td>
<td>The eye is likely damaged beyond meaningful functional repair</td>
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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.34) 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 EAbstract 3045), and others recently have taken a related approach to the problem.35 Such modeling will serve as a rapid, inexpensive method for testing various protective eyewear solutions that might mitigate blast damage to the eye.

Acknowledgments

The authors thank Frank Scribbick and Catherine Fischl of the Ophthalmology Department of University of Texas Health Science Center, San Antonio, Texas, for providing the histopathologic analysis. Presented in part at the annual meeting of the Association for Research in Vision and Ophthalmology, Seattle, Washington, May 2013.

Supported by the Department of Defense Vision Research Program, Award Number W81XWH-12-2-0055. The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Department of the Army or the Department of Defense. William E. Sponsel is a PI with the Australian Research Council Centre of Excellence in Vision Science (ACEV5).

Disclosure: D. Sherwood, None; W.E. Sponsel, None; B.J. Lund, None; W. Gray, None; R. Watson, None; S.L. Groth, None; K. Thoe, None; R.D. Glickman, None; M.A. Reilly, None

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