Experimental evidence to understand mechanical cause of retinal detachment following blunt trauma

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Abstract

**Purpose**: This study aimed to perform an *in vitro* experiment to simulate retinal detachment caused by blunt impact, and provide experimental evidence to understand mechanical causes of traumatic retinal detachment.

**Methods**: The experiment was conducted on twenty-two fresh porcine eyes using a bespoke pendulum testing device at two energy levels (0.1J for low energy and 1.0J for high energy). Dynamic mechanical responses to the impact were examined, including intraocular pressure changes~~, impact force~~ and energy absorption. Another set of thirty-four eyes underwent pathological examination immediately after being subjected to blunt impact. Twelve additional intact eyes were examined as controls. All pathological sections were scored to indicate whether retinal detachment had occurred.

**Results:** A dynamic variation in intraocular pressure was detected following impact and exhibited an approximate sinusoidal oscillation-attenuation profile. The positive and peaks of intraocular pressure were 149.4 ± 18.9 kPa and -10.9 ± 7.2 kPa at low-energy level, and 274.5 ± 55.2 kPa and -35.7 ± 23.7 kPa at high-energy level, showing significant differences (*p* < 0.001 for both levels). Retinal detachments were observed in clearly damaged eyes while only few damages were found in control eyes. The occurrence rate of retinal detachment differed significantly (*p* < 0.05) between the high- and low-energy impact groups.

**Conclusions:** This study provided experimental evidence that shockwaves produced by blunt trauma break the force equilibrium and lead to the oscillation and negative pressure, which mainly contribute to traumatic retinal detachment.

1. Introduction

The retina is a light-sensitive layer of tissue that serves to translate the focused image into neural impulses to the brain. Normally, it is attached to the supporting tissue on the back wall of the eye. When an eye suffers a strong blunt blow (directly or indirectly), a separation of the inner layers of the retina from the underlying retinal pigment epithelium could occur, forming traumatic retinal detachment (TrRD).[1, 2] TrRDs account for 10~40% of all retinal detachments,[3] and are common in children and young people who frequently engage in sports and outdoor activities with high risk of ocular or head trauma, such as diving, boxing, ball games and bungee jumping.[3-9] Abundant clinical evidence indicated that almost all of TrRDs are rhegmatogenous, which occur due to breaks in the retina allowing fluid to pass from the vitreous space into the subretinal space.[10] However, an immediate diagnosis of the TrRD is not always accurate since most clinical signs remain undetected for several weeks or months following detachment.[1, 11] for this reason, understanding the causes of blunt-induced TrRD is important for making a more accurate diagnosis before clinical symptoms develop.

When an eye is subjected to a blunt blow, the globe experiences dynamic deformation in four stages and within a few milliseconds: compression, decompression, overshooting and oscillations,[12] and the associated biomechanical response to the blow may cause TrRD. For example, asynchronous relative motion between the retina and the adjacent vitreous produces a traction that can be large enough to peel the retina away from the supporting tissue.[3, 10, 13] The modeling simulation of blunt eye trauma, presented in a clinical report, suggested that shockwave propagation but vitreous traction is mainly responsible for retinal lesions.[14] It is also believed that negative pressure caused by a blunt blow lead to high strain rates that may tear and detach the retina. Similarly, our previous study, based on a full eyeball model, indicated that the shockwave is specific to retinal break while the negative pressure in the vitreous cavity acting on the retina may directly cause a detachment.[13] These studies focused on simulation using finite element method (FEM), and little experimental evidence exist to support the results of the simulation studies.

In the current study, we developed a bespoke pendulum testing device to produce blunt impacts that may cause TrRD. Mechanical responses to the impact including the globe deformation and the dynamic intraocular pressure (IOP) changes were synchronously collected during the blunt trauma. pathological examination was also performed to detect and observe retinal detachment under different impact energy levels. Our results indicated that the oscillation and negative intraocular pressure following blunt impact mainly contributed to TrRD. The findings would help to understand the mechanism of TrRD from the perspective of biomechanics.

1. Methods
   1. Specimens

All specimens in this study were fresh porcine eyes of animals aged about 6 months, tested within 4 hours post-mortem. To keep the specimens fresh, the eyes were kept in moist storage at 4 ℃ during the 2-hour transportation from a local abattoir. Before the experiment, the peribulbar soft tissue was removed to expose the globe and the optic nerve. Ethical permission for using the eyes in the study was obtained from the local research and ethics committee. The study also adhered to the tenets of the Declaration of Helsinki and the ARVO Statement for the Use of Animals in Ophthalmic and Visual Research.

* 1. Experimental setup



Figure 1 Experimental setup. A bespoke pendulum impact testing device was used to perform the *in vitro* experiment and simulate blunt eye trauma. A spherical hammer mounted at the end of a height-adjustable pendulum rod provided impact with variable energy. A load cell and a high-speed camera were used to record the impact force and the globe’s deformation, respectively. A thin needle was inserted into the vitreous cavity, and was connected to a hydraulic pressure sensor and a bespoke injector using a silicone tube filled with a 10% saline solution. The pressure sensor was used to record dynamic variations in IOP; and the injector was used to maintain static intraocular pressure during blunt trauma. The eye specimen was placed in a 3D printed orbit-shaped holder; and the space between the eye and the holder was filled with gelatin.

The experimental eye impact tests were conducted on fresh porcine eyes (n = 22) using a bespoke pendulum device, including a hammer unit, a pressure measurement unit, an orbit-shaped holder and a high-speed camera system (Figure 1). The hammer part included a spherical hammer (20mm diameter), an adjustable pendulum rod (600mm long in this test), and a load cell (1051V3, Dytran Instruments Inc, USA). The pressure measurement unit consisted of a thin needle, a bespoke injector, a water valve, thick-walled tubes, and a hydraulic pressure sensor (CJGR-15, Xi’an CET Co., Inc, China). The orbit-shaped holder was made from 3D printed polycarbonate. The three-dimensional (3D) model of the orbit was constructed based on CT image sequences from a normal skull of a 28-year-old male subject. Written consent was given from the subject for using his CT images in this study. The 3D reconstruction of the orbit was performed using Mimics 10.01 (Materialise NV Inc., Belgian). A high-speed camera (i-Speed3, OLYMPUS Industrial. Inc, UK) with 100mm macro lens (F2.8 MACRO, TOKINA. Inc, Japan) was used to monitor the eye deformation while being impacted by the hammer. Images were recorded at 1000 frames per second in a pixel resolution of 1280 × 1024.

* 1. Experimental procedure

Before the test, the specimens were placed into the orbit-shaped holder. The space between the eye and the holder was filled with gelatin to simulate viscoelastic fat tissue.[15] We did not consider the extraocular muscles due to earlier evidence that the muscles had little influence on the ocular dynamic response to blunt impact.[15] The holder with the porcine eye was fixed on a platform where the hammer was exactly in touch with the anterior cornea. A thin needle with a tube filled with a 10% saline solution was carefully inserted through the optic nerve into the eye. The injector and the pressure sensor were connected to the same tube. The test started with using the injector to provide a specific IOP of 18 mmHg. Two levels of impact energy were considered as initial conditions in the experiment, involving a low-energy level (0.1 J, n = 12) and a high-energy level (1.0 J, n = 10). The energy was determined by the set height of the hammer, which was linked to the angle between the vertical axis and the pendulum rod, given as:

where E is the impact energy, is the equivalent mass of the hammer and the pendulum rod (106.6 g), is the equivalent radius (568.3 mm) around the center of rotation, is the gravitational acceleration.

During the test, the blunt eye impact was recorded on serials images by the high-speed camera. The images were processed using Pro-Analyst codes (OLYMPUS Industrial. Inc., UK) and used to determine important moments (e.g. the onset and end of the impact) and variations in the hammer speed during eye impact. Impact forces produced by blunt blows were recorded by the load cell at a frequency of 1000 Hz. Synchronously, dynamic IOP changes were recorded by the pressure sensor at a frequency of 1000 Hz.

* 1. Histological examination

We performed histological examinations to characterize retinal pathological changes caused by blunt impact. The examination involved 34 intact eyes (control group n = 12, low-energy impact group n = 11, high-energy impact group n = 11), which were mounted into the orbit-shaped holder without needle insertion. After being subjected to blunt impact, each eye was placed in a FAS eyeball fixative solution (acetic acid, formaldehyde, normal saline and absolute ethanol) until all tissues were completely fixed. Afer fixation, specimens were transported to the ocular pathology laboratory for embedding, sectioning, plating and macroscopic assessment. Sections were prepared by slicing the tissue off the eyeball along a transverse plane. An experienced ophthalmologist scored all sections using 0 or 1 to indicate whether retinal detachment could be detected – whereas 0 meant no retinal injury, 1 meant detection of retinal injury was possible.

* 1. Data analysis

Statistical analyses were performed with MATLAB software (MathWorks. Inc, Natick, USA). All experimental and histological data were subjected to Shapiro-Wilk normality test. Student's t test and Permutation test were used for normally and non-normally distributed data, respectively. We used an alpha level of p<0.05 to determine statistical significance in experimental data. Because of the disperse and hierarchical data, significant differences were analyzed using Mann-Whitney U test for histological data.

1. Results
   1. Impact stage

According to a previous report [12], we divided the blunt eye trauma into four distinct stages: contact, compression, separation and rebound (Figure 2). The contact occurred when the hammer first contacted the anterior surface of the cornea and before any globe deformation (Figure 2a). Compression occurred in the period between contact and the point of reaching the minimum axial length (Figure 2b). Then separation took place when the hammer separated from the globe (Figure 2c), and rebound referred to the stage in which the hammer left away from the specimen and would never be in contact with the eyeball (Figure 2d). Based on this division, we examined mechanical responses (e.g., energy absorption, IOP changes) and pathological changes following low-energy and high-energy blunt impacts.

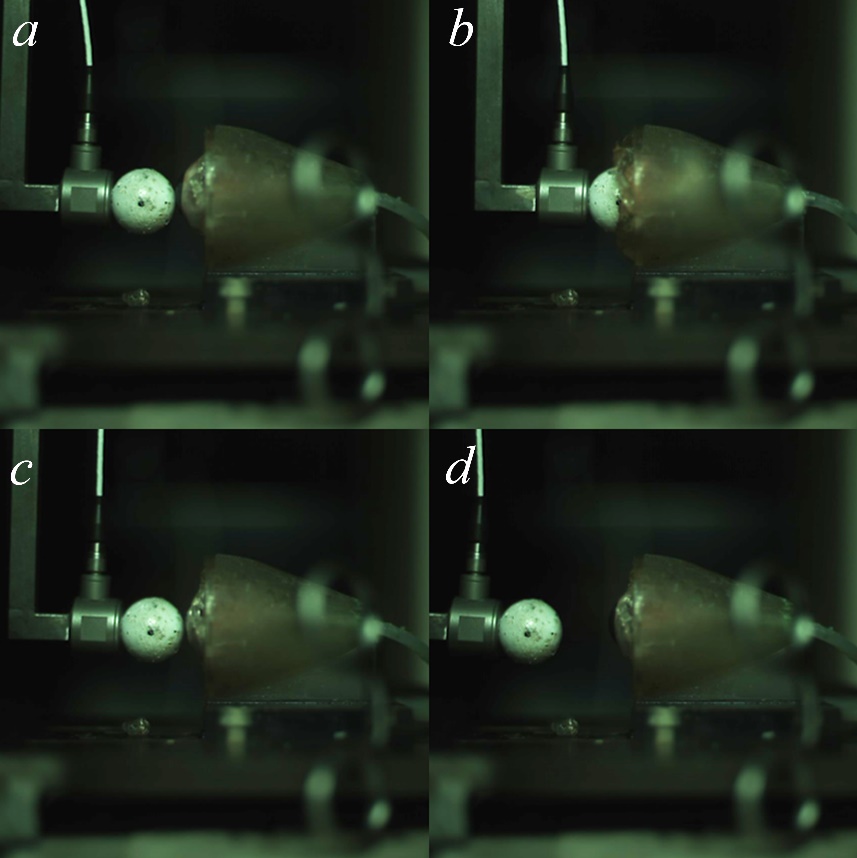


Figure 2 Photographs illustrating the process of a blunt strike on an eye. (a) The hammer just contacted with the anterior surface of the eye. (b) The eye was compressed until the minimum axial length was reached. (c) The hammer just left the surface of the eye. (d) The hammer absolutely left the eye.

* 1. Mechanical responses

The impact forces recorded in tests with two impact energy levels exhibited similar trends (Figure 3a). The peak was observed at the time when the globe was compressed to the greatest extent, followed by a rapid drop. We further examined the peaks of impact force at the two energy levels and found a significant difference between them (12.9±1.9 N at low-energy level and 34.8±9.8 N at high-energy level, p < 0.001), Figure 3b. We calculated the impact energy absorbed by the globe using a kinetic energy equation based on the hypothesis that all energy absorption derives from the kinetic energy loss of the hammer during blunt impact. The calculation indicates that the energy absorption at the low-energy (0.069 ± 0.008 J) and high-energy (0.5 ± 0.17 J) impact levels were significantly different (p < 0.001). The globe’s compression led to a dynamic variation in IOP, exhibiting an approximate sinusoidal oscillation-attenuation profile (Figure 4). Positive components of the IOP were observed at both impact levels while negative components were rarely observed at the low-energy impact level. We further examined the peak values of the positive and negative components, respectively. The results indicate that the positive peaks of IOP were 149.4 ± 18.9 kPa (low-energy level) and 274.5 ± 55.2 kPa (high-energy level), which were significantly different (p < 0.001). A significant difference (p < 0.001) was also found between the negative peaks (-10.9 ± 7.2 kPa at low-energy level and -35.7 ± 23.7 at high-energy level), Figure 4b.



Figure 3 Variations and peaks of impact force under two impact energy levels. (a) Variations of impact force applied on eyes under high- and low-energy levels. The bold lines indicate means; the color bands indicate standard deviations. (b) Peaks of impact force under high- and low-energy levels. \*\*\* p < 0.001.



Figure 4 Variations and positive & negative peaks of dynamic IOP under two impact energy levels. (a) Variations of dynamic IOP in response to blunt impact under high- and low-energy levels. The bold lines indicate means; the color bands indicate standard deviations. (b) Positive and negative peaks of dynamic IOP under high- and low-energy levels. \*\*\* p < 0.001.

* 1. Pathological changes

We examined pathological features of retinal detachment in the control and tested eyes and presented them from three perspectives (Figure 5). Retinal detachment was manifested in post-test pathology by the neurosensory retina being separated from retinal pigment epithelium (Figure 5c). Retinal damage around the optic disc was observed in most sections except for few specimens of the control group. We speculated that retinal damage in the control group could be a pathological artifact because the retina was probably subjected to injury after death or during the sectioning process[16]. We counted the number of retinal detachments observed in sections and found that the rate of retinal damages exhibited a marked increase when specimens were subjected to blunt impact (Table 1). Further, we compared the occurrence rate of retinal detachment under different test conditions and found that there were significant differences in retinal detachment (p < 0.001) between the control (no impact) and impacted (high-energy and low-energy levels) eyes (Figure 6).



Figure 5 Representative Photomicrographs showing (a) no retinal detachment from an eye without any impact, (b) a minute retinal detachment from an eye subjected to low-energy impact and (c) an easily-recognized retinal detachment in an eye subjected to high-energy impact.



Figure 6 The occurrence rate of retinal detachment under no-, low- and high-energy impact levels.

Table 1 Number and proportion of retinal detachment observations in sections obtained from eyes in control, low-energy impact and high-energy impact groups

|  |  |  |
| --- | --- | --- |
| sections | Sum of detached retina scores | Proportion of retinal detachment |
| Control  (n = 33) | 5 | 15% |
| Low-energy Level  (n = 33) | 23 | 70% |
| High-energy Level  (n = 33) | 31 | 94% |

1. Discussion

Retinal detachment is a common injury resulting from blunt ocular trauma. Due to the limitation of experimental setups and conditions, FEM simulation has become the main research tool to represent the mechanical response of the eye to trauma, and predict the incidence of injury. For example, Stitzel et al. developed (and experimentally validated) the well-known Virginia Tech-Wake Forest University (VT-WFU) eye model to predict blunt-induced globe ruptures [17]. Based on this eye model, simulations were run with a variety of impact objects (e.g. baseball, BB, paintball, foam, plastic rods) to understand the effects of the projectile’s characteristics (size, mass, geometry, velocity) on the eye’s response and determine the stress and pressure thresholds for globe ruptures[18]. Additionally, the effects of orbital geometry and increasing lens stiffness on eye injuries were explored using similar FEM simulations [19].

Regarding retinal damage, Rossi et al reported a vitreous- and aqueous-filled eye model to clarify whether blunt trauma shockwave propagation may cause macular and peripheral retinal lesions[14]. They also used this model to predict retinal damage following blast primary effect from a high exposition.[20]. Blast loading is a supersonic pressure or shock wave, which generates high stresses and can lead to intraocular-tissue injuries.[21] Weaver et al. developed an eye model integrated with Lagrangian-Eulerian algorithm to reproduce the retinal damage caused by blast-wave propagation and revealed the protective role of orbital structure in inhibiting the wave propagation[22]. Retinal hemorrhages are common consequences of blast waves followed by abusive head trauma.[23] Finite element eye models based on infant's anatomical structures were constructed to understand the mechanisms of retinal hemorrhages from shaken events[24], present mechanical dynamic response to repetitive shaking loads[25] and determine the retinal forces[25] and vitreoretinal forces[26] contributing to retinal hemorrhages in abusive head trauma. Based on the findings of these simulation studies, the main causes of retinal damage following eye trauma include the excessive deformation of the globe, the shock wave propagating in the intraocular tissues, asynchronous motions between the retina and surrounding tissues, and rapid change of IOP loading on the retina. Although the eye models referred to above were validated using retinal dynamic response to impact loads, obtained in vivo, experimental validation of the models was rarely conducted in earlier studies.

In the current study, we performed an impact experiment to retrieve dynamic responses and pathological changes caused by blunt strikes and examine their relations. It is, to our best knowledge, the first time that experimental evidence was provided to understand the mechanism of TrRD caused by blunt impact. Commonly, TrRD occurs under two physical conditions: 1) retinal break and 2) liquid vitreous passing through the break into the subretinal space[10]. Previous study indicated that shockwave propagation in the eye probably leads to intraocular tissue injuries located at the place where geometrical and physical properties differ significantly.[27] Therefore, retinal breaks were commonly observed along the path of shockwave propagation in clinical and experimental reports[13, 27]. We also observed shockwaves produced by blunt impact, represented by oscillations of intraocular pressure (Figure 4). Different from simulation reports,[14, 20] our experimental results indicated that the shockwaves were rapidly attenuated by the viscous vitreous.

The negative pressure in the eyeball, however, was considered to be mainly responsible for the TrRD. Simulation studies supported that negative pressure caused by blunt trauma is great enough to peel the retina away from the adjacent tissues[13]. Yet, up to now, no experimental evidence has been provided to indicate whether there is the negative pressure counteracting adhesive force of the retinal pigment epithelium leading to TrRD. In the current study, we observed a significant negative pressure in the vitreous cavity when the *in vitro* porcine eye was subjected to high-energy blunt impact. The negative pressure occurred in the overshoot phase, in which the compressed eye recoiled to its original shape (Figure 4a). In the meanwhile, the vitreous immediately flowed forward and filled up the regained anterior space, leading to a negative pressure in the posterior vitreous cavity. The peaks of negative pressure are -10.9 ± 7.2 kPa under low-energy impact and -35.7 ± 23.7 under high-energy impact, both of which were far greater than the retinal adhesive force per unit area (340 Pa).[13] It is, therefore, reasonable to explain that negative pressure following a blunt stroke can produce tensile loading on the retina, forming a transient detachment. If there is no retinal break, the detached retina may adhere to the supporting tissue after the blunt trauma.[10] Retinal breaks make a difference in the occurrence of TrRD in clinical symptoms. This is because liquefied vitreous body can flow in the subretinal space to block retinal adhesion. Blunt trauma is considered as a common cause that leads to vitreous liquefaction, since it tends to accelerate the degradation of a collagen network of vitreous body.[10] To further illustrate the effect of blunt impact on retinal detachment, we performed pathological evaluation after eyes were subjected to blunt trauma. We observed a significant difference in the occurrence rate of TrRD between the experimental and control groups, and also between high- and low-energy impact groups (Figure 6). It suggests that negative pressure is strongly associated with the occurrence of TrRD. It should be addressed that some detachments probably come into being in the process of sample sectioning since the retinal adhesion is weak. In spite of this, the negative pressure in the vitreous further weakens the connection between the retina and its supporting tissue, leading to a higher occurrence rate of TrRD under the high-energy impact.

The current study present experimental evidence to understand mechanical causes of TrRD. Yet, there would be some limitations that need to be addressed. First, the pathological examination of specimens was subjective though a professional and experienced ophthalmologist evaluated all sections with rigorous observation. Another limitation is that the specimens were porcine eyeballs that were extracted from fresh pigs. We rarely observed retinal damages caused by blunt impact because the retina was of good ductility and toughness. In the future, we will perform *in vivo* experiment to give supplementary evidence for the mechanism of TrRD.

In the current study, we performed a *vitro* experiment on blunt eye trauma to understand mechanical causes of TrRD. The results indicated that attenuated oscillations mainly contribute to the retinal break and significant negative pressure in the eye is strongly associated with retinal detachment, which is also supported by the pathological examination. This experimental evidence would help to understand mechanical causes of TrRD following blunt trauma.

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