Mechanical Evaluation of Retinal Damage Associated With Blunt Craniomaxillofacial Trauma: A Simulation Analysis

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Purpose: To evaluate retinal damage as the result of craniomaxillofacial trauma and explain its pathogenic mechanism using finite element (FE) simulation.

Methods: Computed tomography (CT) images of an adult man were obtained to construct a FE skull model. A FE skin model was built to cover the outer surface of the skull model. A previously validated FE right eye model was symmetrically copied to create a FE left eye model, and both eye models were assembled to the skull model. An orbital fat model was developed to fill the space between the eye models and the skull model. Simulations of a ball-shaped object striking the frontal bone, temporal bone, brow, and cheekbones were performed, and the resulting absorption of the impact energy, intraocular pressure (IOP), and strains on the macula and ora serrata were analyzed to evaluate retinal injuries.

Results: Strain was concentrated in the macular regions (0.18 in average) of both eyes when the frontal bone was struck. The peak strain on the macula of the struck-side eye was higher than that of the other eye (>100%) when the temporal bone was struck, whereas there was little difference (<10%) between the two eyes when the brow and cheekbones were struck. Correlation analysis showed that the retinal strain time histories were highly correlated with the IOP time histories (r > 0.8 and P = 0.000 in all simulation cases).

Conclusions: The risk of retinal damage is variable in craniomaxillofacial trauma depending on the struck region, and the damage is highly related to IOP variation caused by indirect blunt eye trauma.

Translational Relevance: This finite element eye model allows us to evaluate and understand the indirect ocular injury mechanisms in craniomaxillofacial trauma for better clinical diagnosis and treatment.

Introduction

Vision impairment can be caused by direct and indirect blunt trauma to the eye.1 In particular, craniomaxillofacial trauma is a type of indirect blunt ocular trauma that is caused by an external force applied to the ocular adnexa and orbital bones, and transmitted to the eye.2 It has been reported that more than 20% of patients with craniomaxillofacial trauma result in severe vision impairment.3

Optic nerve damage is a common reason for vision impairment in craniomaxillofacial trauma.4 When an external force applies to the ocular adnexa and orbital bones, it may indirectly fracture the optic canal, thus displacing, compressing, or intruding the optic nerve, and then damaging the optic nerve.4-6 Vision impairment following craniomaxillofacial trauma is the result of different causes involving commotio retinae, retinal hemorrhage, and retinal detachment.7-10 In clinical practice, retinal injury
without craniomaxillofacial and orbital fractures can easily be overlooked. Evaluation of retinal damage associated with craniomaxillofacial trauma is important to better understand its pathogenic mechanism and improve its diagnosis and treatment.

Finite element (FE) simulation has been commonly used as a robust tool to effectively evaluate ocular injuries in various mechanical conditions, including globe rupture directly caused by a BB (a standard ball bullet with a diameter of 4.5 mm and mass of 0.375 g) projectile, retinal damage following blunt strike and primary blast, and retinal hemorrhage in shaken baby syndrome. Huempfner et al. built a skull model, not including eyeball, to simulate a fist-like object striking a man’s forehead and investigate optic nerve damage. Up to now, many models have been used to evaluate ocular injuries, while few of these models have both the eye and the skull. To evaluate indirect blunt trauma to the eye, it is important to develop a FE model that includes both the eyes and the supporting parts, such as the fat, skull, or skin. As far as we know, such model has not been used to analyze retinal damage caused by craniomaxillofacial trauma.

In this paper, simulations of a solid ball striking different craniomaxillofacial regions were performed to evaluate the resulting retinal damage using a realistic FE model. The model includes the left and right eyes, the skull, the skin, and the orbital fat. The aim of the present study is to evaluate retinal damage caused by craniomaxillofacial trauma and explain its pathogenic mechanism.

Materials and Methods

Case Report

A case report is provided to give a typical example of a severe retinal damage without craniomaxillofacial and orbital fractures after craniomaxillofacial trauma, which could be easily missed in clinical practice. In this case, a 23-year-old Asian male patient was brought to the ophthalmology emergency room at the Peking University 3rd Hospital Eye Center and reported abrupt vision loss after being struck on the forehead by a football 5 days before. There was nothing remarkable in his ocular history prior to the incident. In the diagnosis, no damage was detected in the ocular adnexa and orbital bones, but an optical coherence tomography (OCT) examination showed a slight retinal hemorrhage, an optic nerve contusion, a macular hole, and commotio retinae near the posterior pole (Fig. 1).

Eye Modeling

A FE model of a human right eye was adapted from our previous study (Figs. 2C, 2D). This validated eye model was used to investigate the mechanism of retinal detachment in blunt trauma by simulating the retina peeling away from the supporting tissue. It was an eye model with an idealized shape in which geometrical dimensions were derived from the anatomical measurements of normal human eyes. The structural modeling was performed using SolidWorks 2010 (Dassault Systemes SolidWorks Corp., Shanghai, China), a three-dimensional (3D) computer-aided design software package. The eye model consisted of the cornea, sclera, lens, aqueous, ciliary, zonules, vitreous, and retina. A hexahedral mesh was generated for each part of the model using FE simulation software (ICEM, ANSYS Inc., Canonsburg, PA) with a total of 26,688 elements and 33,621 nodes. The retina was defined as a linear elastic material with an elastic modulus of 20 kPa and a density of 1.1 g/cm³ based on the literature. The material properties of the other ocular tissues were taken directly from published data (Table 1). The FE eye model was validated by matching simulations to the TV-WFU eye model under different impact conditions. Other details of the eye model can be found in our previous publications.

Skull and Skin Modeling

X-ray computed tomography (CT) image sequences of a human skull were obtained from a healthy adult male (age 28 years old, height 173 cm, weight 60 kg) who had no past traumatic damage. Informed consent was obtained from the subject. The study adhered to the tenets of the Declaration of Helsinki.

The CT scan (1 mm contiguous slicing, Siemens Volume Zoom Plus, Siemens Ltd., Munich, Germany)
was performed in the Beijing Tongren Hospital Capital Medical University. The image sequences were imported into image processing software (Mimics 10.01, Materialise Inc., Leuven, Belgium) for 3D reconstruction. The reconstructed model was imported into Geomagic Studio 12 (Geomagic Inc., Frankfurt, Germany) for smoothing. The skull model was split in half along the sagittal plane. The left half of the skull model was removed and replaced with a mirror duplicate of the right half to form a symmetrical skull model. Structures that are unlikely to play any significant role in the mechanism of ocular injury in craniomaxillofacial trauma, such as the ear bones and the oral cavity, were excluded in the skull model. A skin model was constructed to cover the outer surface of the skull model with a thickness of 1 mm. The skull model and the skin model were imported into FE simulation software. Triangular shell elements were created to discretize the models with a total of 57,612 elements and 28,810 nodes (Fig. 2A). The material models and parameters of the skull and skin are listed in Table 1.

**Model Assembling and Fat Modeling**

The FE eye model and the FE skull model were assembled according to a reported method (Fig. 2). First, the lateral eye protrusion (LP), lateral

<table>
<thead>
<tr>
<th>Parts</th>
<th>Material Model</th>
<th>Material Parameters</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cornea</td>
<td>Nonlinear elastic</td>
<td>Stress-strain relation, $u = 0.47$</td>
<td>Union et al.²⁵</td>
</tr>
<tr>
<td>Sclera</td>
<td>Nonlinear elastic</td>
<td>Stress-strain relation, $u = 0.47$</td>
<td>Union et al.²⁵</td>
</tr>
<tr>
<td>Lens</td>
<td>Linear elastic</td>
<td>$E = 6.88$ MPa, $u = 0.42$</td>
<td>Czygan et al.²²</td>
</tr>
<tr>
<td>Zonules</td>
<td>Linear elastic</td>
<td>$E = 357.78$ MPa, $u = 0.42$</td>
<td>Czyan et al.²²</td>
</tr>
<tr>
<td>Ciliary</td>
<td>Linear elastic</td>
<td>$E = 11$ MPa, $u = 0.47$</td>
<td>Power et al.³⁰</td>
</tr>
<tr>
<td>Retina</td>
<td>Linear elastic</td>
<td>$E = 20$ kPa, $u = 0.49$</td>
<td>Power et al.³⁰</td>
</tr>
<tr>
<td>Aqueous</td>
<td>Liquid</td>
<td>Shock EOS linear</td>
<td>Power et al.³⁰</td>
</tr>
<tr>
<td>Vitreous</td>
<td>Viscoelastic</td>
<td>$C_1 = 1530$ m/s, $s_1 = 2.1057$</td>
<td>Lee et al.²⁴</td>
</tr>
<tr>
<td>Fat</td>
<td>Viscoelastic</td>
<td>$G_0 = 0.9$ kPa, $G_\infty = 0.5$ kPa, $\beta = 14.26$ s⁻¹, $K = 2.0$ GPa</td>
<td>Schoemaker et al.²¹</td>
</tr>
<tr>
<td>Skull</td>
<td>Linear elastic</td>
<td>$E = 14.5$ GPa, $u = 0.35$</td>
<td>Robbins and Wood²²</td>
</tr>
<tr>
<td>Skin</td>
<td>Linear elastic</td>
<td>$E = 1.0$ MPa, $u = 0.45$</td>
<td>Larrabee and Sutton³³</td>
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</tbody>
</table>

$E$, elastic modulus; $u$, Poisson’s ratio; $K$, bulk modulus; $G_0$, initial shear modulus; $G_\infty$, infinite shear modulus; $\beta$, viscoelastic decay constant; $C_1$, speed of sound through the material; $s_1$, the coefficient related to the speed of the shocked material.
distance (LD), superior eye protrusion (SP), and superior distance (SD) were measured in the CT images; orbital width (OW) and height (OH) were measured in the reconstructed skull model. Then, the measurements were used as a guide to rotate and translate the eye model to the correct position in relation to the skull model. The right eye model was symmetrically copied to create a left eye model. The bilateral orbital space unoccupied by the eye models was filled with a fat model (Fig. 2B). The material model and parameters of the orbital fat model are listed in Table 1.

Simulation

The impactor was simplified as a ball with a diameter of 5 cm, a density of 1 g/cm³ and a Young’s modulus of 9800 MPa. The material parameters of the impactor were the same as those of human bone. The initial velocity of the impactor was set to 5 m/s, which was similar to a normal man’s hand speed.

The impactor produced kinetic energy of 0.82 J. The absorbed impact energy was considered to be the kinetic energy reduction after collision, which is expressed as:

$$E_p = \frac{1}{2}m(v_i^2 - v_r^2)$$

where $E_p$ is the energy absorbed by the head, $m$ is the mass of the impactor, $v_i$ is the initial velocity, and $v_r$ is the rebound velocity of the impactor.

Four regions were considered in the simulation: the frontal bone (case 1, Fig. 3A), the temporal bone (case 2, Fig. 3B), the brow (case 3, Fig. 3C), and the cheekbones (case 4, Fig. 3D).

<table>
<thead>
<tr>
<th>Strike Region</th>
<th>$C_t$ (ms)</th>
<th>$v_i$ (m/s)</th>
<th>$v_r$ (m/s)</th>
<th>$E_p$ (J)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 1</td>
<td>2.30</td>
<td>5.00</td>
<td>1.82</td>
<td>0.71</td>
</tr>
<tr>
<td>Case 2</td>
<td>3.05</td>
<td>5.00</td>
<td>3.06</td>
<td>0.51</td>
</tr>
<tr>
<td>Case 3</td>
<td>2.45</td>
<td>5.00</td>
<td>2.58</td>
<td>0.60</td>
</tr>
<tr>
<td>Case 4</td>
<td>4.65</td>
<td>5.00</td>
<td>3.85</td>
<td>0.33</td>
</tr>
</tbody>
</table>

CT, collision time; $v_i$, impact velocity; $v_r$, rebound velocity; $E_p$, energy absorption.

Statistical Analysis

Pearson correlation tests were performed to assess the relationship between the peak IOP (pressure of the vitreous) and peak retinal strain time histories of the struck-side eye using SPSS 13.0 (SPSS Inc., Chicago, IL). It should be noted that the locations of the peak strain on the retina and peak pressure of the vitreous varied at different times.

Results

Simulations indicated that the energy absorption was different when the head was struck at different craniomaxillofacial regions (Table 2). The energy of 0.71 J (86.8% of the total kinetic energy) was absorbed when the frontal bone was struck (case 1), whereas only 0.33 J was absorbed when the strike was...
on the cheekbones (case 4). The absorbed energy in case 1 was more than twice that in case 4.

Figure 4 shows the dynamic IOPs in two eyes when the temporal bone was struck (case 2). As the shockwave propagated, the IOP exhibited dramatic variations and vibrations from the struck-side eye to the other (Fig. 4A). The IOP vibration continued for some time (Fig. 4B) after the collision finished.

Figure 5 shows the strain distribution in the retina when the macular strain reached the peak. The location of the macula is indicated by an arrow in each subfigure. Equivalent strain was used to evaluate retinal damage. Figures 6 and 7 show the retinal strain time histories on the macula when strike occurred on different craniomaxillofacial regions: (A) strike on the frontal bone; (B) strike on the temporal bone; (C) strike on the brow; and (D) strike on the cheekbones.
histories on the macula and the ora serrata, respectively, in four cases. The transmitted force produced a strong retinal strain variation on the macula, followed by a prolonged attenuation. The peak strains on the ora serrata were higher than those on the macula in all four cases. A relatively high peak strain on the ora serrata (0.27 on the struck-side eye) was found when the cheekbones were struck (case 4). The strike on the frontal bone (case 1) produced a higher peak strain (~0.18) on the macula of both eyes. However, a relatively low peak strain (~0.12) on the macula was found for strikes on the brow and cheekbones (cases 3 and 4, respectively). The macular strain exhibited a great difference when the temporal region was struck (case 2). In that case, the macular peak strain of the struck-side eye (0.20) was 122.2% higher than that of the other eye (0.09). To provide a clearer illustration, the peak strains on the macula, ora serrata, and their relative differences are listed in Table 3. Importantly, the 95th percentile was used as the definition of the peak value to reduce the outliers resulting from possible numerical artifacts.

Table 3. The Peak Strains on the Macula, the Ora Serrata, and Their Relative Differences of the Struck-Side Eye and the Opposite-Side Eye When Craniomaxillofacial Trauma Occurred in Different Regions

<table>
<thead>
<tr>
<th>PSM</th>
<th>PSO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eye</td>
<td>SS</td>
</tr>
<tr>
<td>Case 1</td>
<td>0.18</td>
</tr>
<tr>
<td>Case 2</td>
<td>0.09</td>
</tr>
<tr>
<td>Case 3</td>
<td>0.12</td>
</tr>
<tr>
<td>Case 4</td>
<td>0.13</td>
</tr>
</tbody>
</table>

RD = (SS eye − OS eye)/OS eye. PSM, peak strains on the macula; PSO, ora serrata; RD, relative differences; SS, struck-side; OS, opposite-side.

Figure 8 shows a comparison of the peak retinal strain time histories and the peak IOP time histories when strike occurred on different craniomaxillofacial regions: (A) strike on the frontal bone; (B) strike on the temporal bone; (C) strike on the brow; and (D) strike on the cheekbones.

Discussion

Craniomaxillofacial trauma is often associated with retinal injuries, such as commotio retinae, retinal hemorrhage, and retinal detachment.7–10 At present, the mechanism of indirect blunt eye trauma is not well understood. Malbran et al.2 proposed that forces spreading to the retina through the ocular adnexa and orbital bones could result in retinal damage. While cavitation or negative pressure caused by outward bending of the orbital wall was also considered as the primary cause of the intraocular tissue injuries,34 our simulation showed that shockwaves produced by the strike, representing a dynamic IOP, were transmitted from the struck-side eye to the other eye (Fig. 4). This finding suggests that shockwave propagation in the eye probably leads to intraocular tissue injuries. Therefore, the tissues tend to be damaged along the path of shockwave propagation, especially at the interface where the geometrical and physical properties differ significantly between the two tissues.35 For this reason, strain concentration were found near the macula when the frontal bone was struck (Fig. 5), since the macula is on the opposite side of the struck position (a contrecoup injury). Correlation analysis indicated that retinal strain was highly correlated with IOP (correlation coefficient >0.8, P = 0.000, Fig. 8). It suggests that IOP could be used as an indicator for retinal injuries caused by direct or indirect eye impacts.
trauma. Recently, dynamic responses of the interior ocular tissues are different to obtained experimentally, since the inner structures in the eyeball are not able to be visualized using high-speed imaging technology. Measurement of IOP using a dynamic pressure transducer that is connected into the eye through a hollow needle and a tube would be useful to evaluate intraocular tissue damage in ex vivo eye impact experiment.

At present, equivalent stress or strain has been accepted as an important criterion for evaluating ocular tissue injuries. Tissue injuries likely occur when the maximal stress or strain exceeds the material damage limits. The simulation result indicated that the retinal strain reached its peak at the ora serrata in the struck-side eye (Table 3), which is consistent with the clinical presentation that retinal dialysis following blunt trauma often occurs around the ora serrata. However, most minor dialyses can spontaneously seal themselves; evaluation of retinal damage in this region is less important than that in the macula. Approximately 10% of traumatic retinal detachment cases are related to macular holes. Retinal damage of the macula could greatly harm vision. As a result, a strike on the frontal bone would cause a higher strain on the macula (Fig. 5), because the macula located on the opposite side of the impact position and is thus prone to stress concentration. In addition, the absorbed kinetic energy was greater for a strike on the frontal bone (86.8% of the total energy) than on the other craniomaxillofacial regions (Table 2). Therefore, the risk of retinal damage in the macular region would be higher when a person experiences frontal bone trauma, as mentioned in the case report. While lower macular strain was found when the cheekbones were struck, the absorbed energy in this case accounted for only 40% of the total impact energy. When a strike occurred on the frontal bone, brow, and cheekbones, the macular strains were nearly equal in the struck-side eye and the opposite-side eye. This finding suggests that fundus examination for both eyes is necessary when the frontal bone, brow, and cheekbones suffer from strikes. The macular strain of the struck-side eye was more than 100% greater than that of the other eye when strike occurred on the temporal bone. This finding indicates that the risk of retinal damage is likely higher in the struck-side eye when a person experiences temporal bone trauma.

The primary limitation of this work is the lack of accurate material properties of the retina used for the FE model of the human eye. The thinness and fragility of the retina make it extremely difficult to determine its material properties by experiment. In the present model, the retina was assumed to be a linear elastic material with a Young’s modulus of 20 kPa, which was determined by an inverse solution from an experiment in which forces were applied to detached retinas. However, soft tissues exhibit significant time-dependent properties. Instead of exhibiting static mechanical behavior, these tissues present a higher stress strength but lower ductility in dynamic conditions. As we lacked the dynamic and nonlinear material parameters of the retina, a static linear material (Young’s modulus of 20 kPa) was used for the retina model instead of dynamic properties. Therefore, the results only indicated a relative high risk of retinal damage (not an absolute damage risk) when strike occurred at a certain craniomaxillofacial region. Another significant limitation is the lack of direct validation for the simulation. In the previous study, we validated the eye model by six matched simulations with the VT-WFU eye model. The validation simulations were conducted with three types of blunt projectiles with two respective velocities: BB (56 and 92 m/s), foam (10 and 30 m/s), and baseball (34.4 and 41.2 m/s). The tissue exhibits similar dynamic responses to the same loading rate. Since the loading rates used in the previous validation cover those in the present simulation, the simulation results in this paper are considered to be reliable although no direct validation was conducted.

In conclusion, our simulation provided retinal dynamic responses to craniomaxillofacial trauma, allowing an evaluation of retinal damage risk. The results suggest that the risk of retinal damage varies greatly when craniomaxillofacial trauma occurs in different regions. Correlation analysis indicated that the retinal strain time histories are highly correlated with the IOP time histories. It suggests that IOP can be used as an indicator for retinal injuries caused by direct or indirect eye trauma. This finding is important for evaluating retinal damage in experimental ex vivo eye impact study.

Acknowledgments

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