EXTENDED REPORT

Computer modelling study of the mechanism of optic nerve injury in blunt trauma

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Aim: The potential causes of the optic nerve injury as a result of blunt object trauma, were investigated using a computer model.

Methods: A finite element model of the eye, the optic nerve, and the orbit with its content was constructed to simulate blunt object trauma. We used a model of the first phalanx of the index finger to represent the blunt body. The trauma was simulated by impacting the blunt body at the surface between the globe and the orbital wall at velocities between 2–5 m/s, and allowing it to penetrate 4–10 mm below the orbital rim.

Results: The impact caused rotations of the globe of up to 5000°/s, lateral velocities of up to 1 m/s, and intraocular pressures (IOP) of over 300 mmHg. The main stress concentration was observed at the insertion of the nerve into the sclera, at the side opposite to the impact.

Conclusions: The results suggest that the most likely mechanisms of injury are rapid rotation and lateral translation of the globe, as well as a dramatic rise in the IOP. The strains calculated in the study should be sufficiently high to cause axonal damage and even the avulsion of the nerve. Finite element computer modelling has therefore provided important insights into a clinical scenario that cannot be replicated in human or animal experiments.

Anterior traumatic optic neuropathy and optic nerve avulsion may result from a blunt injury where a foreign object intrudes between the globe and the orbital wall.^{1–3} Although often caused by apparently moderate trauma, optic nerve damage has devastating consequences for the victim, resulting in partial or permanent loss of vision in the affected eye. Some optic nerve injuries occur with only minor injury to the globe, which suggests a minimal amount of contact between the eye and the foreign object, and which implies that the mechanism of the associated optic nerve damage is highly specific. The exact biomechanical mechanisms of injury are not clear. Forceful rotation of the globe by the blunt object, anterior movement of the globe displaced by the foreign object, and a sudden rise in the intraocular pressure (IOP), have all been mentioned as possible optic nerve injury mechanisms.⁴ Although all of these hypotheses appear to have a sound basis in clinical findings and trauma histories, they remain at the level of speculation because of lack of appropriate experimental validation.

Computer models are increasingly proving to be an alternative to complicated, practically unfeasible, or unethical (human or animal) experiments. The finite element method, which is a mathematical method for solving complex physical problems on domains with complicated geometries, is commonly used in constructing such computer models. In ocular trauma, finite element models of the isolated globe have already been used to simulate injury caused by airbag inflation, and projectile impacts.^{5–8} We have developed a more complete finite element model which involves the eye, the optic nerve, and the orbit with its contents with the aim of investigating high acceleration, and impact type of trauma. In recent papers,^{9–10} which dealt with the eye trauma in head impacts of various severity, we discussed possible scenarios and mechanisms that can result in the evulsion of both the globe and the optic nerve. In this study we examine possible causes of the optic nerve injury caused by collision with a blunt object. A number of different blunt objects have been implicated in patients with optic nerve avulsion, including

snooker cues, golf clubs, and paintballs.^{2–3–11} However, finger poking seems to be one of the most common causes of injury,^{1–12} and this was the reason to choose a finger to represent the blunt body in this study.

METHODS

Finite element model

The finite element model of the eye and the orbit is shown in figure 1. The geometry of the orbit is a simplified representation of a realistic geometry obtained from the magnetic resonance imaging (MRI) scans of an adult volunteer. The globe is a sphere of 12 mm in radius with a 0.8 mm thick outer membrane shell representing the corneoscleral shell. An opening, 1.5 mm in radius, in the scleral shell at the posterior pole where the optic nerve is inserted is covered by a 0.25 mm thick membrane¹³ which represents the lamina cribrosa. The inner space of the globe is occupied by a viscoelastic tissue representing the vitreous. A small portion of the intraocular space (less than 10 mm³ in volume) near the anterior pole of the globe was modelled as a lumped volume compartment with a pressure-volume characteristic of the water. We used an algorithm controlling the influx and outflux of volume into this compartment¹⁴ to simulate aqueous humour production/absorption in the anterior chamber, and to set the IOP at 15 mmHg initially (see the appendix for more details). The volume of the compartment was kept constant during the impact simulation. The orbital space is filled with a soft viscoelastic solid representing fat. The globe is submerged into the fat up to 4 mm (one third of the radius) over the equator. The geometry of the extraocular muscles was taken from Robinson.¹⁵ Each of the muscles was composed of five one dimensional Hill-type elements with passive and active components in parallel. A passive pre-stress of 0.05N (approximately 5 g) and an active tone of 0.1N were prescribed to the rectus muscles. The active tone was kept

Abbreviation: IOP, intraocular pressure

Figure 1 Finite element model. (A) Eye orbit and the blunt body (finger). (B) Globe, optic nerve, and the extraocular muscles.

constant during the simulations. The muscles were not in contact with any of the model components except for the globe. The optic nerve sheath was represented by a tubular membrane shell of 1.5 mm cross sectional radius of the same thickness and material properties as the sclera. The nerve was filled with a soft, nearly incompressible, solid representing nervous tissue. The path of the optic nerve was chosen in such a way that the length of the nerve was 8 mm longer than the distance between the apex of the orbit and the posterior pole of the globe (see fig 1B). All the components of the eye orbit except for the muscles were meshed together. The decision to connect the globe to the fat was based on the results of our previous computer and experimental studies, where different types of connection were explored.^{9 10 16} Low shear stiffness of the fat allows relatively easy rotation of the globe with respect to the orbit.

The first phalanx of the index finger was modelled as a 30 mm long cylinder with an ellipsoidal tip. The cross section was elliptical with the short axis of 6 mm and long axis of 8 mm. The digital bone was represented by a rigid circular cylinder of 3 mm radius. The fingernail, represented by a 0.8 mm thick shell, stretched 15 mm from the tip, and covered an arc of 45°. We modelled the skin as a 1 mm thick membrane.

The material models and physical properties of the tissues represented in the model are given in the appendix. All of the solids in the model were meshed with eight noded brick elements, and all the shells/membranes with quadrilateral elements. In total, 55 320 elements and 49 900 nodes were required to construct the model. Modelling and meshing were performed in ANSYS 8.1 (Ansys Inc, Canonsburg, PA, USA).

Simulations

Our aim was to simulate blunt injury occurring from relatively moderate trauma, such as a sports accident. Basketball related injuries would be a typical example.¹² We took reports of trauma from the literature to estimate critical parameters such as impact velocity, but we did not attempt to

Figure 2 Deformed mesh at the moment where the maximal penetration of the blunt body is reached (5 ms). (A) Three dimensional view. (B) Cross sectional view in the horizontal plane passing through the centre of the globe.

reconstruct any particular traumatic event. Rather, we explored simplified, plausible scenarios. We chose the position and orientation of the finger so that the impact on the globe itself was minimised. Hence, the axis of the finger was midway between the orbital rim and the globe, and oriented towards the apex of the orbit. The finger was initially moving in the direction of its axis at a velocity between 2–5 m/s, which is typical for rapid hand movements.¹⁷ Throughout the impact the orbital bone was restrained from moving, so that the initial velocity of the blunt body was also the impact velocity relative to the eye orbit. At the moment when the finger came into contact with the surface of the orbital fat, a triangular deceleration pulse was prescribed to the digital bone to bring it to rest after the finger has travelled 6–10 mm below the orbital rim. Relatively small penetration is consistent with our aim of simulating optic nerve injury that occurs without direct contact between the foreign body and the nerve. The shape and intensity of the deceleration pulses were consistent with those measured in head impacts upon a fall from a small height,^{18 19} or the impact of a small object to the head.²⁰

We simulated four impact locations: two in the horizontal plane passing through the centre of the globe; one on the medial side and one on the lateral side; and two in the vertical plane of symmetry, one on the superior and one on the inferior side. We used the LS-DYNA (Livermore Software Technology Corporation, Livermore, CA, USA) commercial finite element package to carry out simulations. This is an explicit finite element code for dynamic simulation that uses reduced integration and hourglass control for solid and shell elements. A penalty based algorithm was used for the contact between the finger and the fat/globe, as well as for the contact between the globe and the extraocular muscles. Sliding without friction was assumed in both cases.

Figure 3 Estimated lateral velocity of the globe (A), estimated rotational velocity of the globe in the horizontal plane (B), and the intraocular pressure (C).

RESULTS

Figures 2–5 apply to the case where the initial finger velocity was 4 m/s, the maximal penetration was 8 mm, and the impact location was in the horizontal plane, at the medial side of the orbit. The deformed mesh at the moment where the maximal penetration of the blunt body is reached ($t = 5$ ms) is shown in figure 2. The globe was displaced laterally, and the anterior pole rotated towards the blunt body. There was a significant dent on the medial side of the globe as a result of the contact with the finger. The globe rotation and lateral displacement reached their peak values approximately at the moment when maximal penetration

Figure 4 Von Mises stress (MPa) in the sclera and the optic nerve shell at $t = 5$ ms. The view is 30° from the horizontal plane, opposite to the impact site.

was achieved. At that moment in time the bulging of the fat surface opposite to the location of impact was the most pronounced. For $t > 5$ ms the deformation of the fat surface was more evenly spread, and the rotation of the globe was smaller. The forces generated in the extraocular muscles were relatively small. In the lateral rectus the peak increase in force was approximately 0.15 N, whereas in the medial rectus the force dropped from the initial pre-stress value. This all suggests that the movement of the globe was to a large degree dictated by the movement of the fat. The displacement of the globe and the optic nerve is illustrated in figure 2B, which shows the deformed mesh in the horizontal plane at $t = 5$ ms. The stretching of the optic nerve at the lateral side, at the point of insertion to the globe, is the result of both the globe rotation and the fact that the anterior part of the optic nerve moves laterally, while the posterior part remains in place.

Motion of the globe

The bulk motion of the globe was quantified through the motion of the axis, which connects the anterior and posterior poles. The translation then refers to movement of the imaginary point at the middle of the axis, and the rotation to the rotation around the same point. The bulk translation was mainly in the mediolateral direction and the rotation was in the horizontal plane. The anterior velocity and displacement of the globe were small. The results for the calculated velocities for lateral translation and for rotation are shown in

Figure 5 Maximal principal strain in the lamina cribrosa at $t = 5$ ms (anterior view).

figure 3. The maximal velocity was around 0.8 m/s and the maximal rotational velocity was somewhat over 4000°/s. The maximal angle of rotation was around 10°.

Stress and IOP

The stress in the sclera and the optic nerve shell at $t = 5$ ms are displayed in figure 4. The main site of stress concentration was in the optic nerve at the insertion into the sclera and opposite to the impact. The other affected site was the edge of the dented region of sclera. The peak strain generated in the most affected region of the optic nerve shell was above 10%. The pressure in the anterior chamber was taken as a reference for the IOP. Elsewhere within the globe the mean normal stress (pressure) was of similar magnitude. The maximal value was close to 300 mm Hg (see fig 3C). This increase in IOP appears to be a direct consequence of the compression of the globe by the finger. In the lamina cribrosa, which is weaker structure than the sclera, increased IOP caused strains of over 20% (see fig 5).

Parameter variation

Variation in the location of impact did not produce significantly different results for the stress intensity and the principal sites of stress concentration (opposite to the site of finger impact). However, this result should be treated with caution because of the geometric idealisation used in the model. Generally speaking, increasing the impact velocity and the depth of penetration caused an increase in the globe rotation and translation, as can be seen in figure 6. The IOP

was sensitive to the increase in the depth of penetration, but not to the impact velocity. For $H = 4$ mm the peak IOP was 180 and for $H = 10$ mm, IOP was 350 mm Hg.

DISCUSSION

The results of our computer simulations are consistent with the location and nature of the blunt body optic nerve injuries reported in the clinical literature.^{1-3,11} The point at which the optic nerve inserts into the sclera, on the opposite side to the finger impact, would appear to be under the greatest strain. Large strains were also calculated in the lamina cribrosa. The results suggest that there are two principal mechanisms of injury: the rotation of the optic nerve relative to the globe, and the increased IOP arising from the deformation of the globe. The angle between the globe and the nerve at the point at which the nerve is inserted into the sclera is affected both by the globe rotation and by the lateral globe translation. Both of these motions seem to be caused principally by the dynamics of the orbital fat, which is displaced by the foreign object, as passive muscle forces appear to be too weak to restrain the movement of the globe significantly. The estimated maximal rotational velocity was up to one order of magnitude larger than that observed in the most rapid saccades of normal eye movements.²¹ Although it is not trivial to quantify the contribution of each of the two suggested mechanisms separately, it seems that the nerve rotation is the dominant mechanism in the optic nerve avulsion; whereas the IOP increase is more likely to cause the damage in the optic nerve head. The anterior motion of the globe, which has been suggested as a potential injury mechanism, was small compared to the other motion components and, based on the current results, it should not be considered as a major cause of the optic nerve damage.

The peak strains obtained from the simulations were within the range of those observed to cause rupture in the cornea and sclera,⁶ which may account for the possibility of the optic nerve avulsion. The strains in the lamina cribrosa were even larger, and they were beyond the levels thought to cause nerve damage in the situations such as glaucoma.²²⁻²⁴ Although criteria derived from the studies on chronically elevated IOP may not apply directly to impact, where the increase in the IOP is massive but of a very short duration, our results suggest that the injury to the optic nerve head in blunt trauma is a very realistic possibility.

Given the range of velocities used in the simulations, it can be said that the study reinforces the belief that seemingly benign incidents can lead to serious ocular injury. It should be said, however, that the motion profiles used in the study are highly idealised, and that much more complex impact motion can be expected in reality. The impact will cause the head to move, and thus affect the velocity of the blunt body relative to the orbit. Generally speaking, modelling of the eye and the orbit is a challenging task and drastic simplifications are inevitable. As the focus was not on the injuries to the eye, we used a relatively simplistic representation of the globe, both in terms of the geometry and in terms of the material properties. This may have significant effect on the results concerning the stresses in the eye shell and the IOP.²⁵ As a crude test of this effect we ran the model with the thickness of the corneoscleral shell varying from 0.5–1.0 mm and with its Young's modulus varying from 1 MPa to 25 MPa. The results were surprisingly consistent in terms of the strains in the lamina cribrosa, which were always around 20%. Also, although the effect on the IOP was significant, its peak value was always above 120 mm Hg. Although this is encouraging, modelling of the globe deserves much more attention in the future. Finally, it should also be mentioned that artefacts of the finite element mesh, such as sharp angles, may somewhat exaggerate stress concentration at those points, which

Figure 6 Variation of the peak lateral velocity (A) and the peak rotational velocity (B) of the globe as a function of the impact velocity and the depth of the blunt body penetration.

is another reason to treat the current results with caution. It is therefore important for us to caution that computer modelling studies should not yet be used medicolegally as a means of estimating the "inflicted force" in reconstruction of individual traumatic scenarios. On the other hand, our study indicates that finite element computer modelling of ocular trauma can provide important insights into precise biomechanical mechanisms of tissue injuries, in situations that cannot be replicated in human or animal experiments. Clinical observations in traumatic optic neuropathy/avulsion correlate with, and provide important qualified validation of, our finite element computer model of the eye and orbit in blunt trauma.

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APPENDIX

MATERIAL PROPERTIES OF THE TISSUES REPRESENTED IN THE MODEL

Following Winters,²⁶ we used an exponential law to express the force-elongation characteristics for the passive component of each muscle, which can be expressed as:

$$F = \kappa \exp [\alpha(\lambda - 1)]$$

where F is the force, λ is the engineering strain, and $\kappa = 3.33 \times 10^{-2}$ N and $\alpha = 13.8$ are constants determined to fit the experimental data reported by Scott.²⁷

To model a single fibre in a muscle, the value of constant κ and the total active force per muscle were divided by the number of fibres in a muscle (five).

Aqueous humour was modelled as fluid with bulk modulus equal to that of water. A lumped parameter algorithm for fluid filled airbags provided by LS-DYNA software was used to simulate aqueous humour in the anterior chamber. The pressure and volume in the chamber are governed by the following equations:

$$m = V\rho - V_0\rho_0$$

$$IOP = K \ln(V_0/V)$$

where V is the volume of the compartment, ρ is density, K is the bulk modulus of the aqueous humour, m is the net mass influx of humour into the chamber, and subscript "0" denotes condition where $IOP = 0$.

The orbital and the finger bone were modelled as rigid bodies. Following the paper by Lee *et al*,²⁸ the vitreous was represented as a four parameter linear viscoelastic material with a creep compliance (J) of the following form:

$$J(t) = J_m + J_k [1 - \exp(-t/t_k)] + t/h_m$$

where t is time, and J_m is instantaneous shear compliance, J_k is retarded shear compliance, t_k is retardation time, and h_m is residual viscosity. The orbital fat tissue, and the subcutaneous tissue of the finger were modelled as linear viscoelastic solids with the time-dependent shear modulus (G) of the form:

$$G(t) = G_i + (G_0 - G_i)e^{-\beta t}$$

The long time shear modulus, G_i , and the short time shear modulus, G_0 , were estimated from the results reported by Shoemaker *et al*²⁹ for different types of fat tissue by assuming a standard viscoelastic solid model. The decay constant β was

Table A Mechanical properties of the tissues			
Tissue	Reference	Material model	Parameter values
Sclera	Belleza <i>et al</i> ²²	Linear elastic	E = 5.5 MPa; ν = 0.47
Vitreous	Lee <i>et al</i> ²⁸	Linear viscoelastic	J_m = 0.1 Pa ⁻¹ ; J_k = 0.4 Pa ⁻¹ ; t_k = 0.27 s; h_k = 3.13Pa s; K = 2100 MPa
Aqueous humour		Fluid	K = 2100 MPa
Nerve	Belleza <i>et al</i> ²²	Linear elastic	E = 5.5 MPa; ν = 0.47
Neural tissue	Sigal <i>et al</i> ²³	Linear elastic	E = 0.03 MPa; ν = 0.499
Lamina cribrosa	Sigal <i>et al</i> ²³	Linear elastic	E = 0.3 MPa; ν = 0.499
Orbital fat	Shoemaker <i>et al</i> ²⁹	Linear viscoelastic	G_1 = 0.5 kPa; G_0 = 0.9 kPa; β = 50 s ⁻¹ ; K = 2100 MPa
Subcutaneous tissue	Shoemaker <i>et al</i> ²⁹	Linear viscoelastic	G_1 = 3 kPa; G_0 = 5 kPa; β = 50 s ⁻¹ ; K = 2100 MPa
Skin	Larrabee and Sutton ³¹	Linear elastic	E = 1 MPa; ν = 0.45
Nail	Wu <i>et al</i> ²²	Linear elastic	E = 170 MPa; ν = 0.3

picked to reflect typical values reported for the soft tissue in the literature.³⁰ All other tissues were modelled as linear elastic and were characterised by Young’s modulus (E) and Poisson’s coefficient (ν). Density of 1000 kg/m³ was assumed for all of the tissues represented in the model. The material models and material constants are summarised in table A.